Arrhythmias

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Arrhythmias

Arrhythmias Part I :

- Definition
- Introduction to Cardiac conduction system.
- Introduction to ECG interpretation .
- Mechanism of Arrhythmias .
- Etiology .
- Presentation .
- Diagnostic approaches

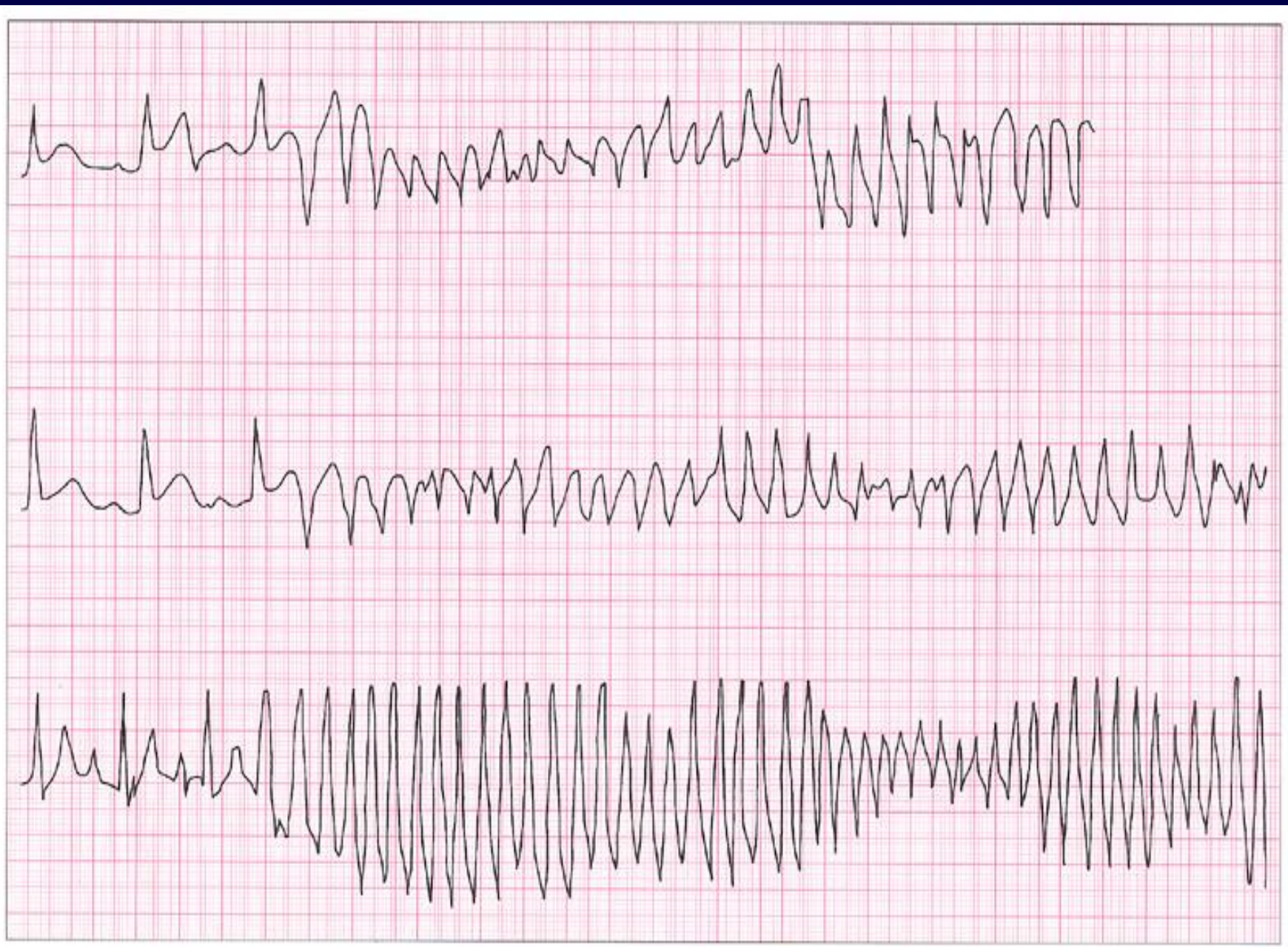
Arrhythmias Part I :

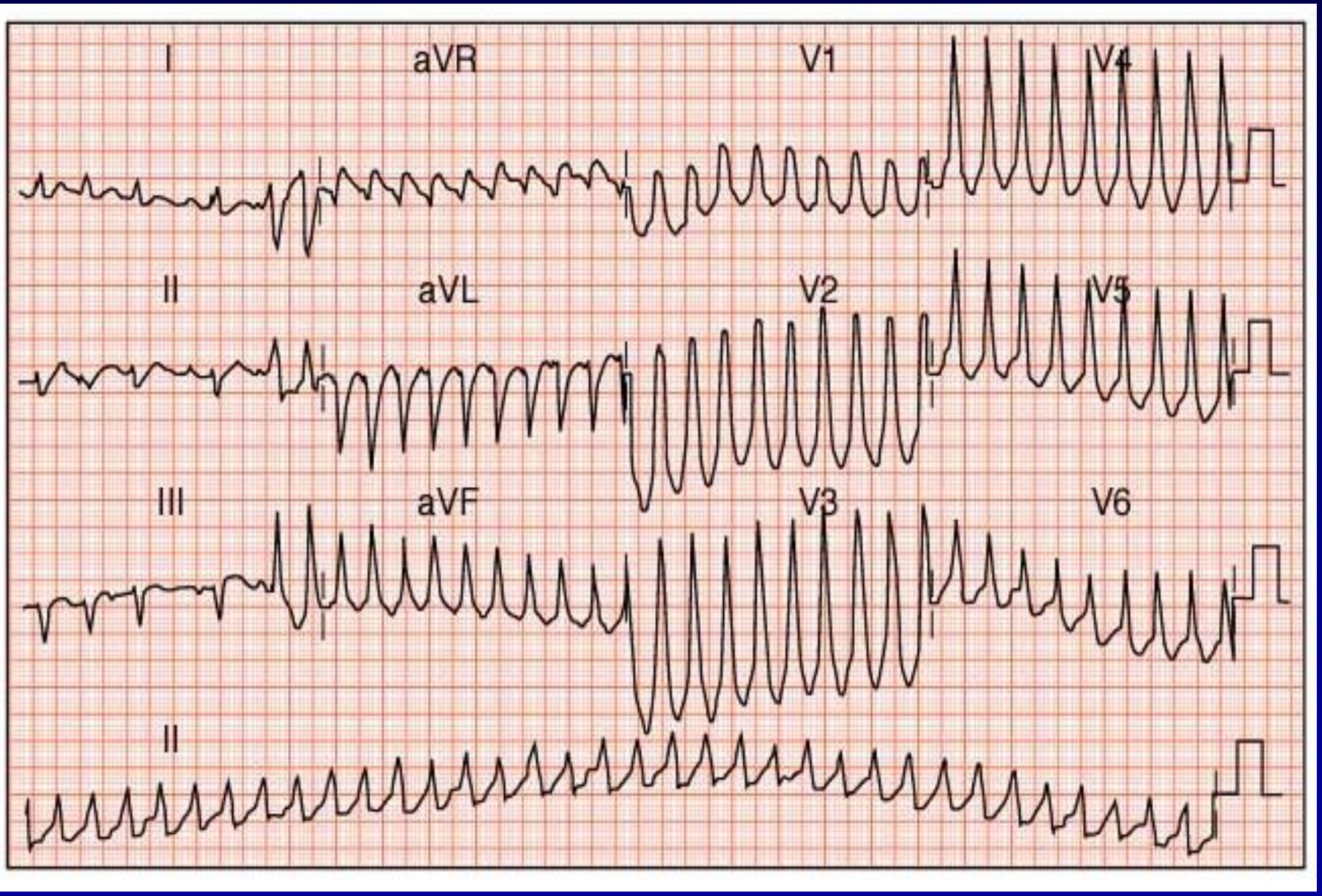
- Classifications and management

Arrhythmias

Definition :

Disorders of the origin, rate, rhythm and conduction of heart electrical activation .

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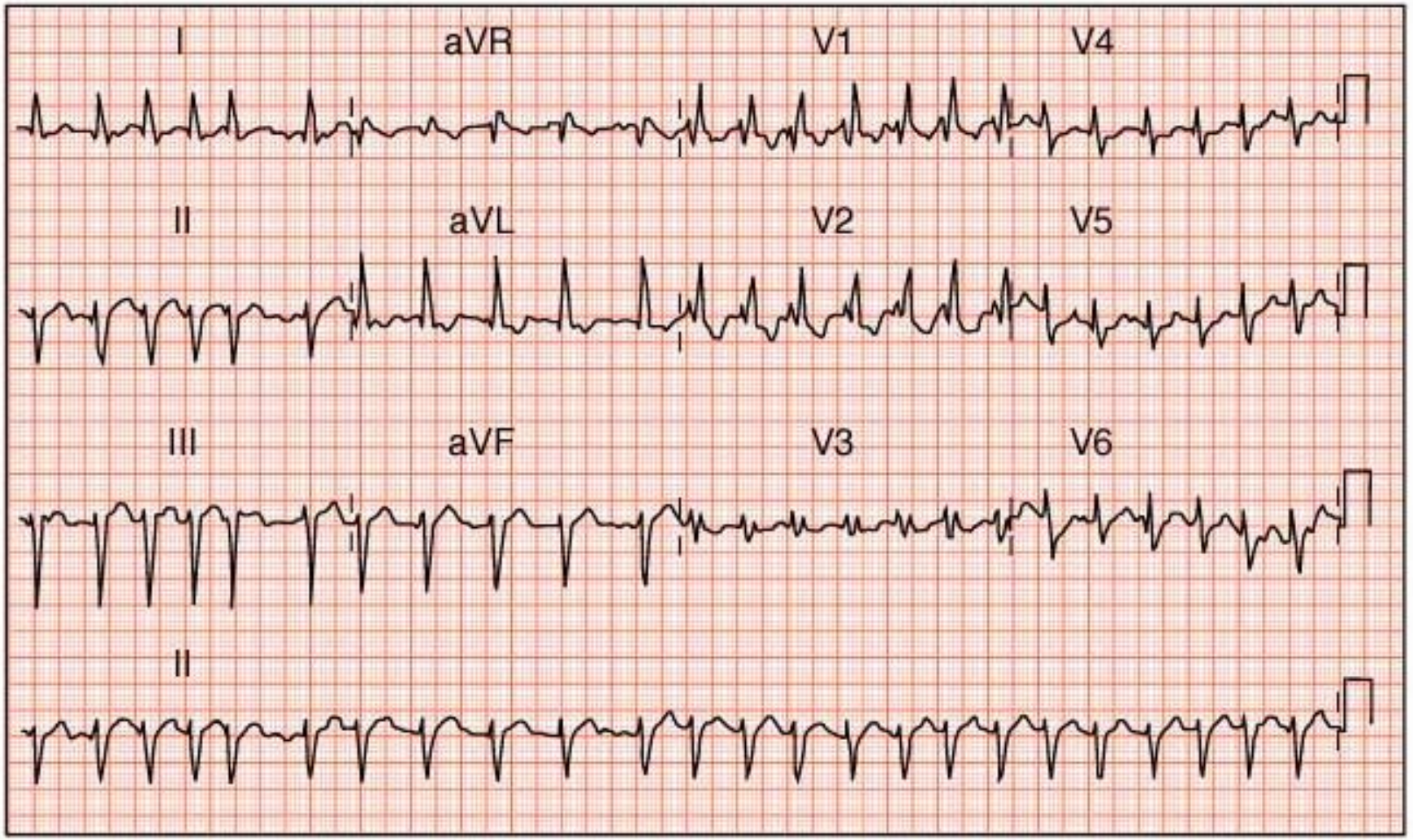
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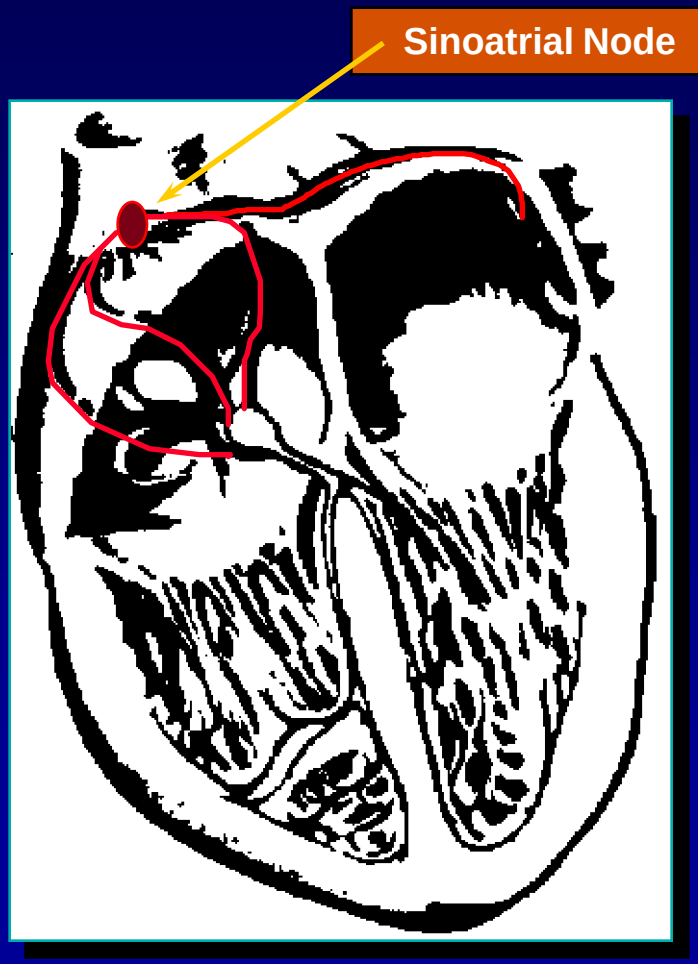
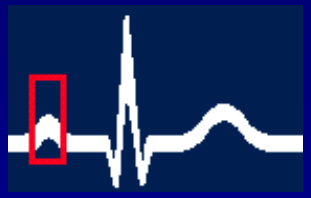
Cardiac cells contract without Nervous Stimulation.

- Most cardiac muscle is contractile (99%), but about 1% of the myocardial cells are specialized to generate action potentials spontaneously.
- These cells are responsible for a unique property of the heart: **its ability to contract without any outside signal.**
- The signal for myocardial contraction comes NOT from the nervous system but from specialized myocardial cells also called **auto rhythmic cells.**
- These cells are also called **pacemaker cells** because they set the rate of the heart beat .

Cardiac Conduction System

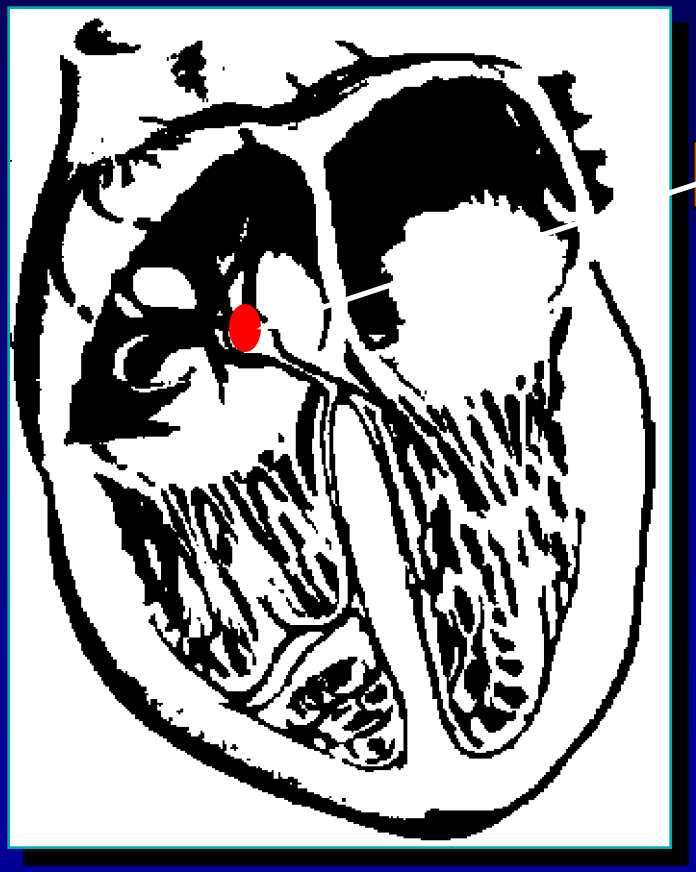
-**Sinoatrial Node :** - Situated at the junction of the SVC and RA It acts as pacemaker ,it Comprises specialized atrial cell ,during normal sinus rhythm ,depolarization wave propagates through the atria via sheet of atrial myocytes to the AV node

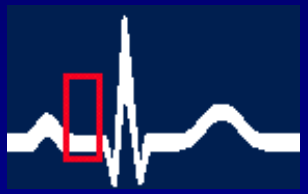




Cardiac Conduction System

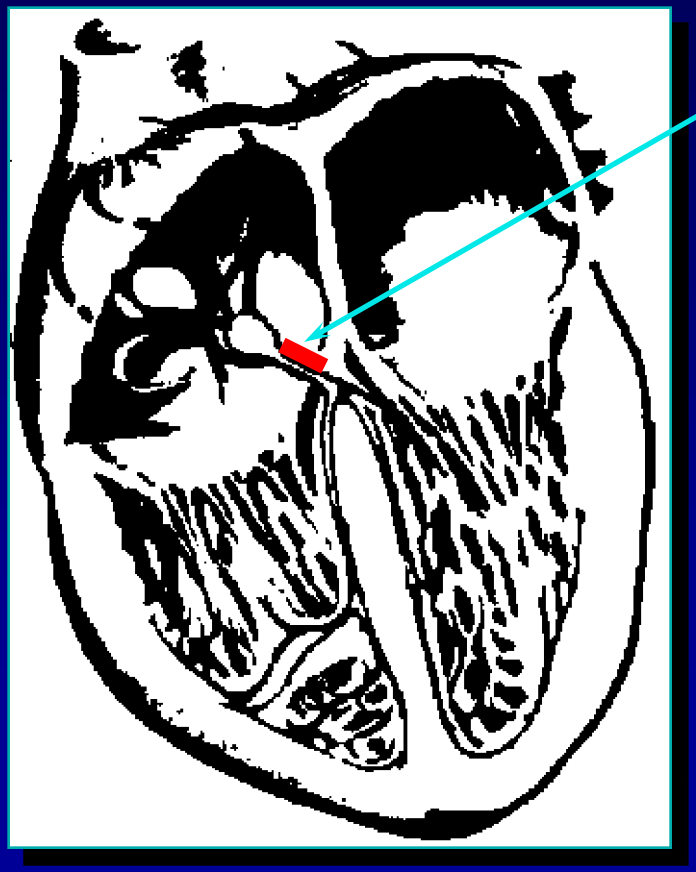
-AV Node : Conducts relatively slowly ,producing a necessary time delay between atrial and ventricular contraction .



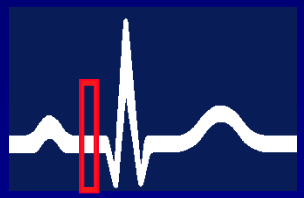


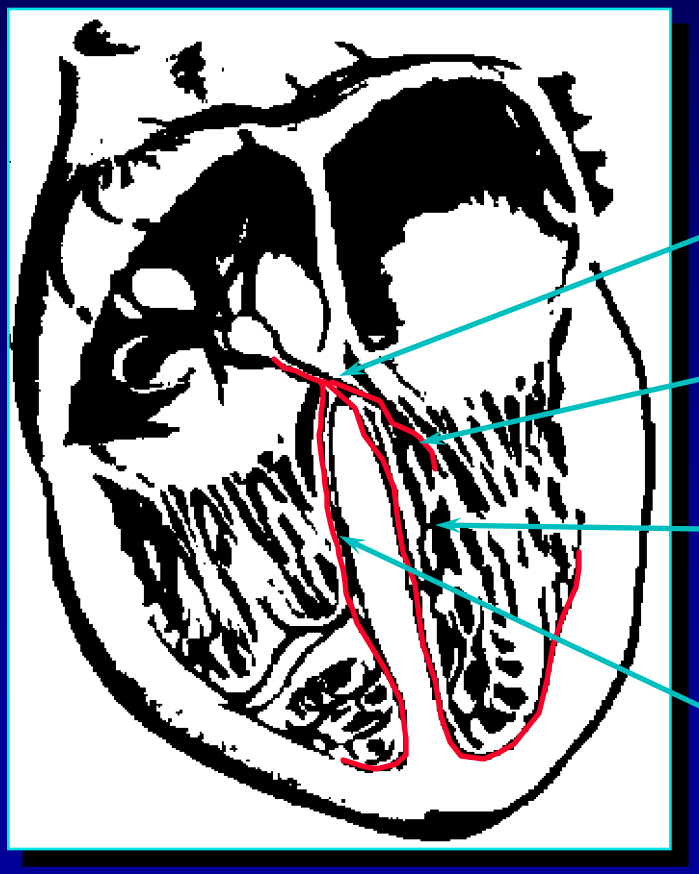
Cardiac Conduction System

-The His-Purkinje system : is comprised of the bundle of His extending from the AV node into the interventricular septum ,Rt and Lt bundle branches .

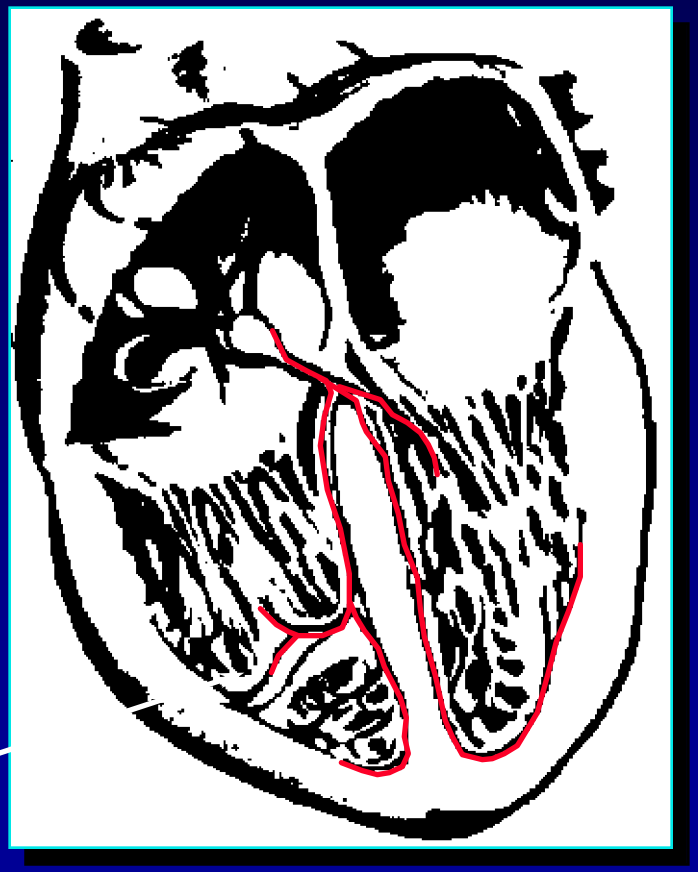


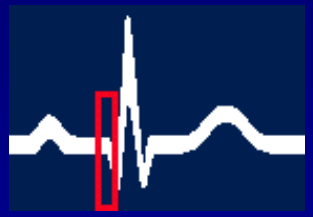
Cardiac Conduction System



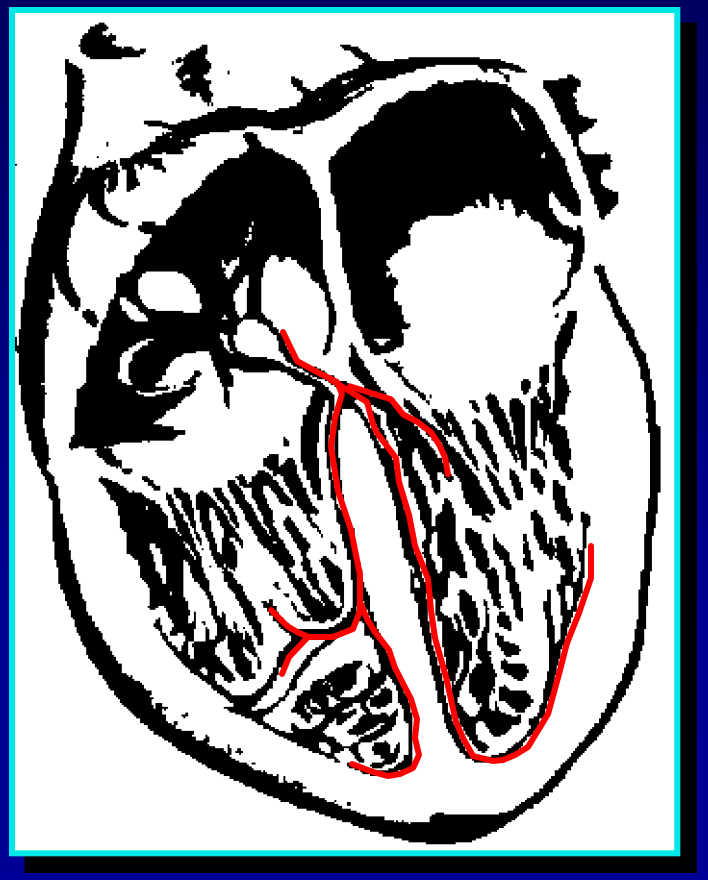


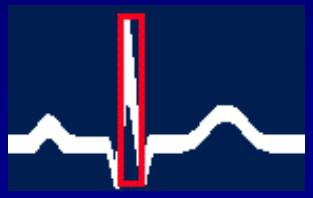
Cardiac Conduction System





Cardiac Conduction System

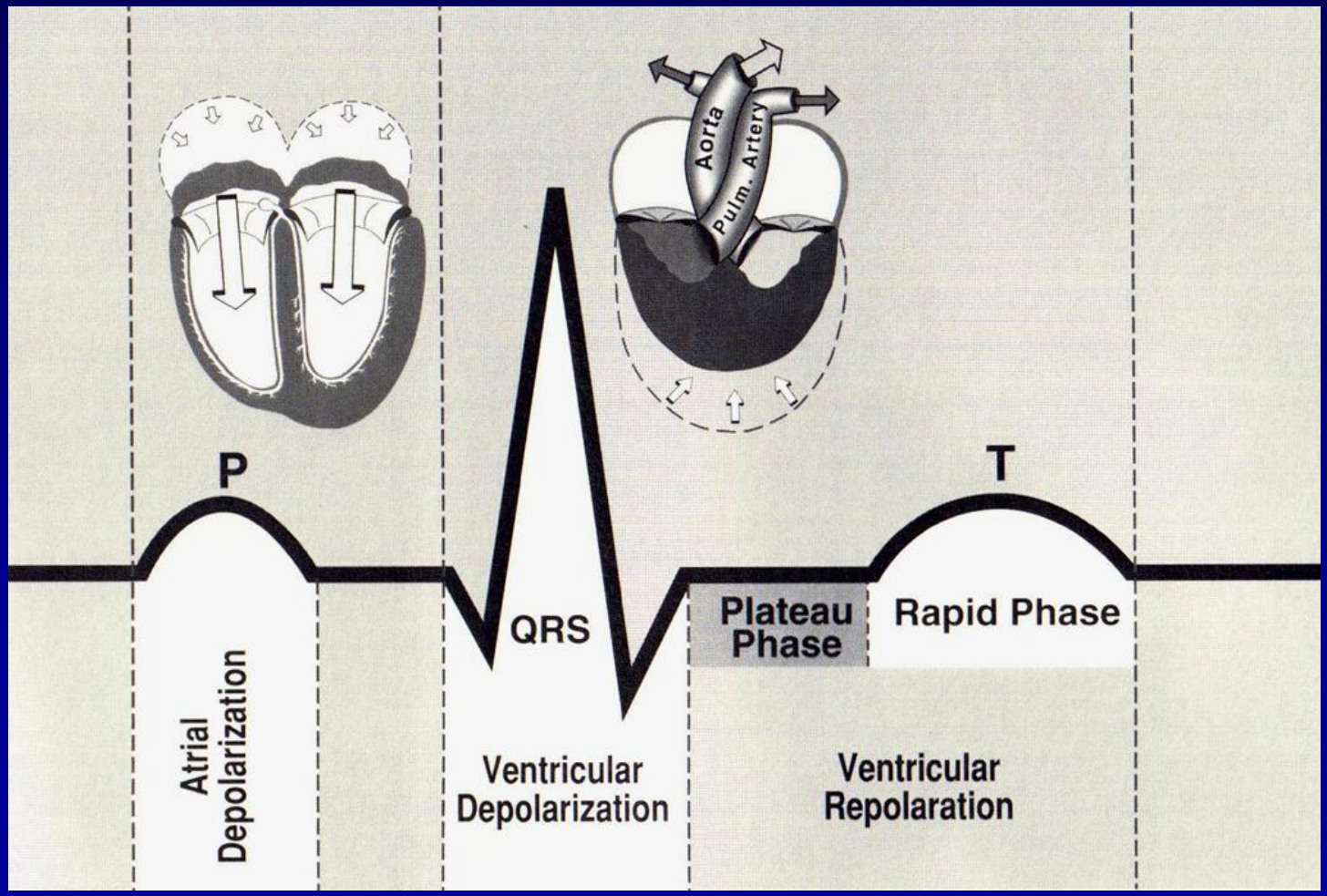




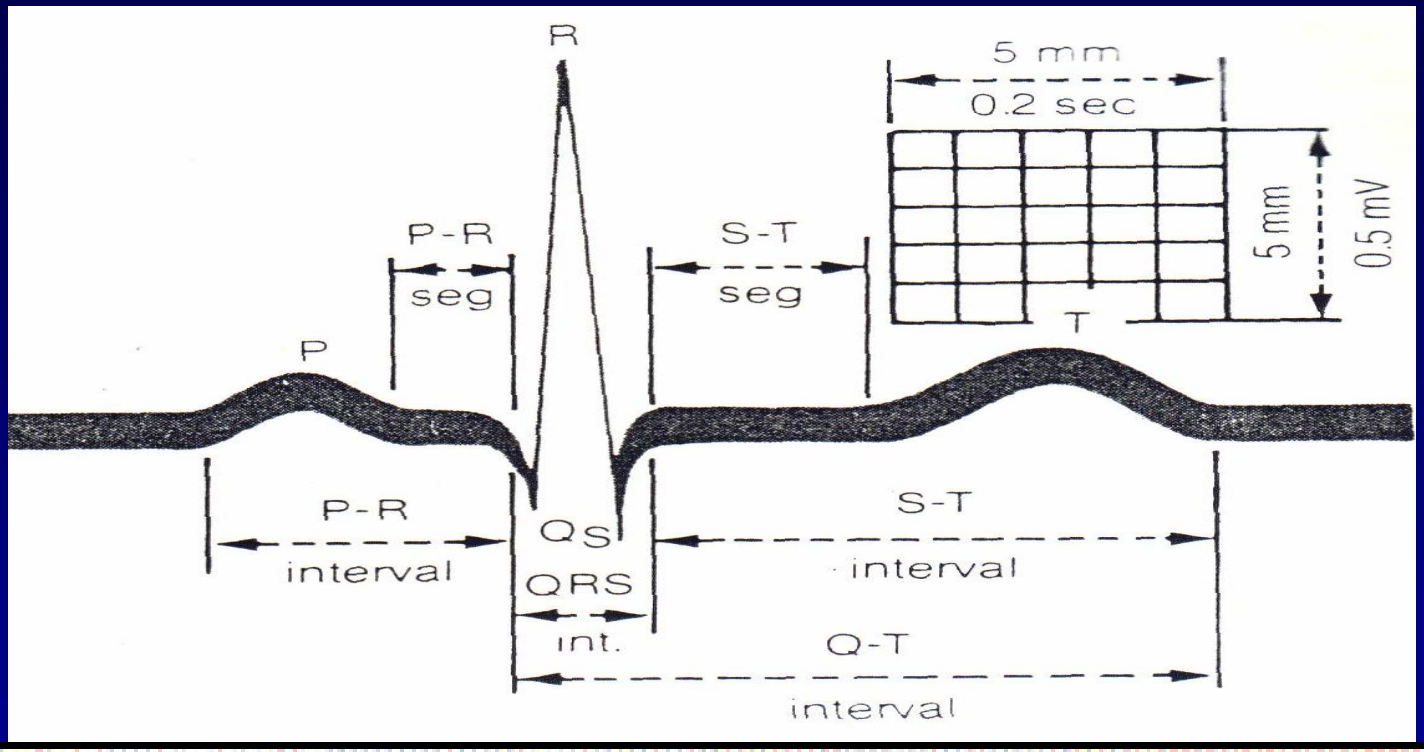
Normal Sinus Rhythm

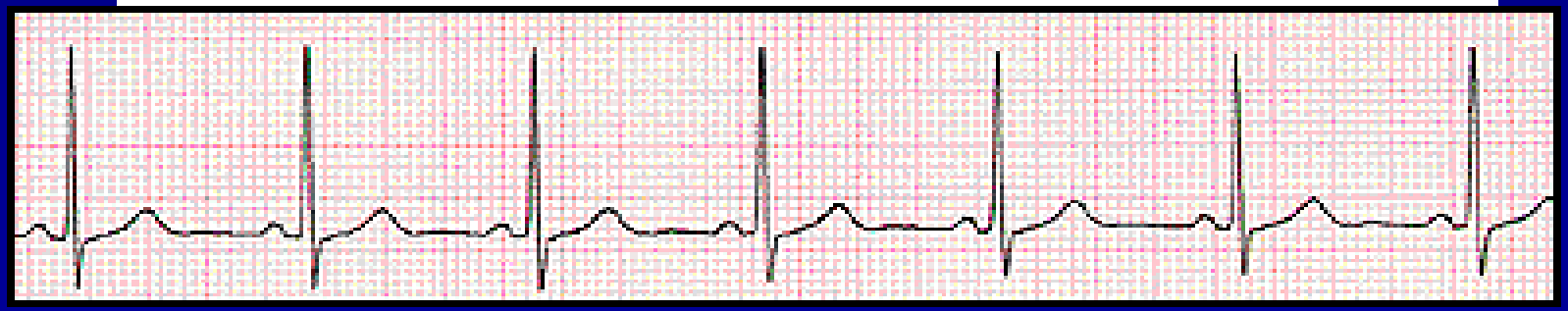
- Normal sinus rhythm produced by SA node .
  - P – WAVE follows by QRS complex in a predictable relationship .
  - All “P” WAVES look alike, all ALL QRS complexes are narrow .
  - R – R interval is regular
  - RATE: 60 – 100 bpm

Normal Sinus Rhythm



Normal Sinus Rhythm





Basic Rhythm Strip Interpretation

1. **Determine the rate**. Does the atrial rate equal the ventricular rate.
2. Is the **rhythm** regular/irregular?
3. Find the **P wave**. Is there a P wave for every QRS?
4. Determine the **PRI** (Normal 0.12-0.20 sec)
5. Find the **QRS** (Normal <0.12seconds)
6. Find the T wave, QT and ST segment .
7. The **axis**
8. Any ectopic beats?

What are the Mechanisms of Arrhythmias??

1- Disorder of impulse formation.

a) Automaticity.

b) Triggered Activity

c) 2- Disorder of impulse conduction.

d) Conduction delay.

e) Re-entry .

3. Combined disorder.

1-Disorder of impulse formation.

A-Automaticity :

Automaticity is the ability of the heart muscle cells to generate an electrical impulse. All cells in the heart have this capacity, but only certain cells, such as those in SA node, are responsible for generating heartbeats , the normal pacemaker.

1- Normal automaticity :

a- Enhanced Normal automaticity: leads to sinus tachycardia .

b- Suppressed Normal automaticity : leads to sinus bradycardia

2- Enhanced abnormal automaticity :

Refers to generation of an electrical impulse from abnormal pacemaker or cells within the myocardium e.g. atrial or ventricular arrhythmias .

B- Triggered activity:

Arrhythmias due to triggered activity are rare; when they occur, they are often due to problems in the ion channels in the heart muscle cell.

Which caused by electrical instability in the myocardial cell membrane.
A typical example of this is **Torsade de Pointes.** They can also occur as a side effect of certain anti-arrhythmic drugs .

2- Disorder of impulse conduction:

A- Conduction delay : e.g. AV block and BBB

B- Re-entry : Re-entrant arrhythmias occur when an electrical impulse recurrently travels in a tight circle within the heart, rather than moving from one end of the heart to the other and then stopping

Etiology

- Physiological
- Pathological:
  - Valvular heart disease.
  - Ischemic heart disease.
  - Hypertensive heart diseases.
  - Congenital heart disease.
  - Cardiomyopathies.
  - Carditis.
  - RV dysplasia.
  - Drug related.
  - Pericarditis.
  - Pulmonary diseases.
  - Others.

Arrhythmia Presentation

- **Asymptomatic**
- **Symptomatic**
  - ✓ **Palpitation.**
  - ✓ **Dizziness.**
  - ✓ **Syncope or near syncope**
  - ✓ **Chest discomfort**
  - ✓ **Fatigue**
  - ✓ **Dyspnea.**
- **Lethal and dangerous presentation**
  - ✓ **Sudden cardiac death.**

Diagnostic approach

1- Medical History.

2- Clinical examination:

A-BP and heart rate .

B- feeling for peripheral pulses

C- Auscultation of the heartbeat with a stethoscope.

Finding related to the underling cardiac cause .

It can give a general indication of the heart rate and whether it is regular or irregular.

3 -Diagnostic test .

A- Electrocardiogram ECG .

B- Holter monitor 24 hours

(is an EKG recorded over a 24-hour period.)

C- Electrophysiology study .

4- Other impotent test to diagnosis the cause :

A- Echocardiography .

B- Coronary angiography .

Antiarrhythmic drugs
Vaughan William classification

class	mechanism	action	notes
I	Na^+ channel blocker	Change the slope of phase 0	Can abolish tachyarrhythmia caused by reentry circuit
II	β blocker	↓heart rate and conduction velocity	Can indirectly alter K and Ca conductance
III	K^+ channel blocker	1. ↑action potential duration (APD) or effective refractory period (ERP). 2. Delay repolarization.	Inhibit reentry tachycardia
IV	Ca^{++} channel blocker	Slowing the rate of rise in phase 4 of SA node(slide 12)	↓conduction velocity in SA and AV node

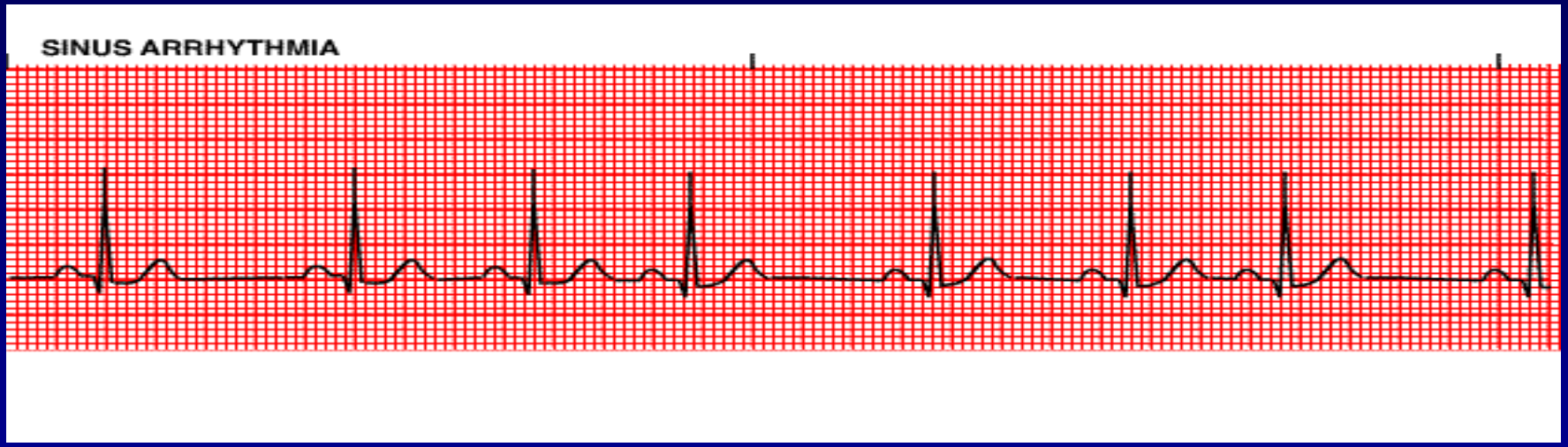
Classification of Arrhythmia

Arrhythmia can be classified according to the rate or the mechanism, but the important classification is according to site of origin:

1. Sinoatrial nodal arrhythmia
2. Atrial arrhythmia
3. Junctional arrhythmia
4. Ventricular arrhythmia
5. Abnormal heart pulse conduction
    1. Atrio-ventricular block
    2. Intra-ventricular block

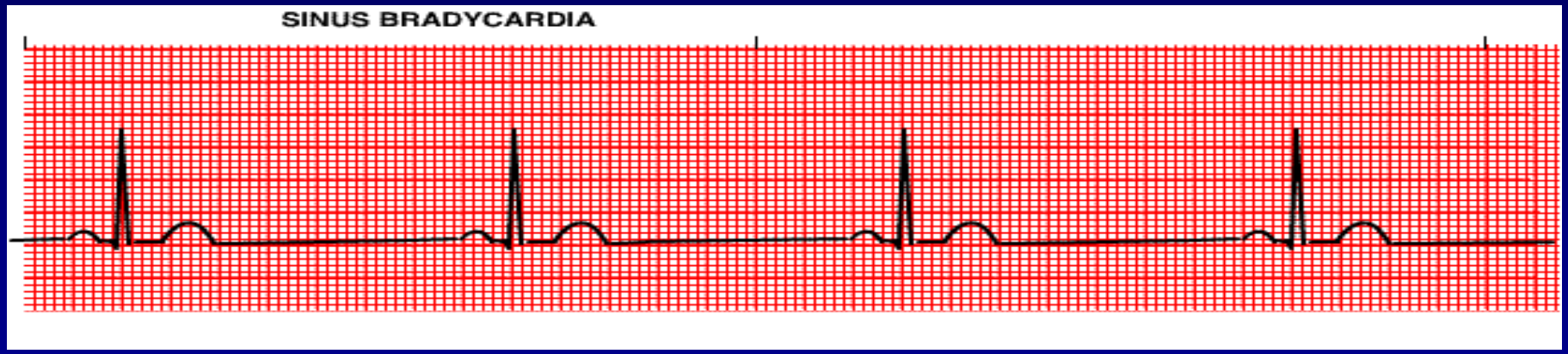
1-Sinoatrial nodal arrhythmia:

- Sinus arrhythmia.
- Sinus bradycardia.
- Sinus tachycardia.
- Sick sinus syndrome.


SINUS ARRHYTHMIA

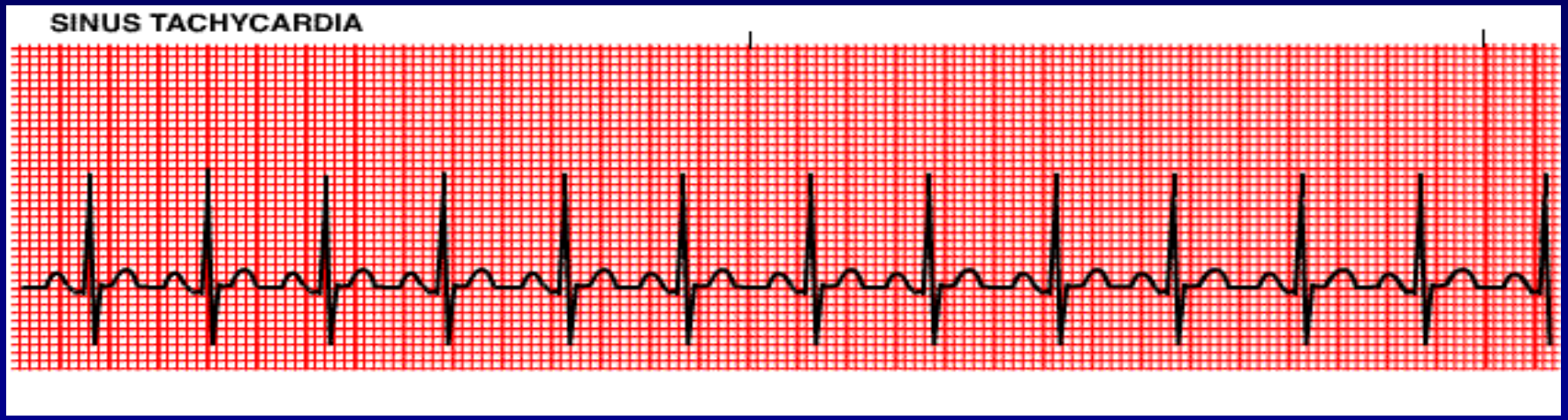
Sinus Arrhythmia

- **P wave:** sinus
- **QRS:** normal
- **Conduction:** normal
- **Rhythm:** regularly irregular
- Alteration of heart rate during respiration ,rate usually increases with inspiration and decreases with expiration.
- This rhythm is most commonly seen with respiration due to fluctuations in vagal tone.
- The non respiratory form is present in diseased hearts and sometimes confused with sinus arrset (also known as "sinus pause").
- Treatment is not usually required unless symptomatic bradycardia is present.


SINUS BRADYCARDIA

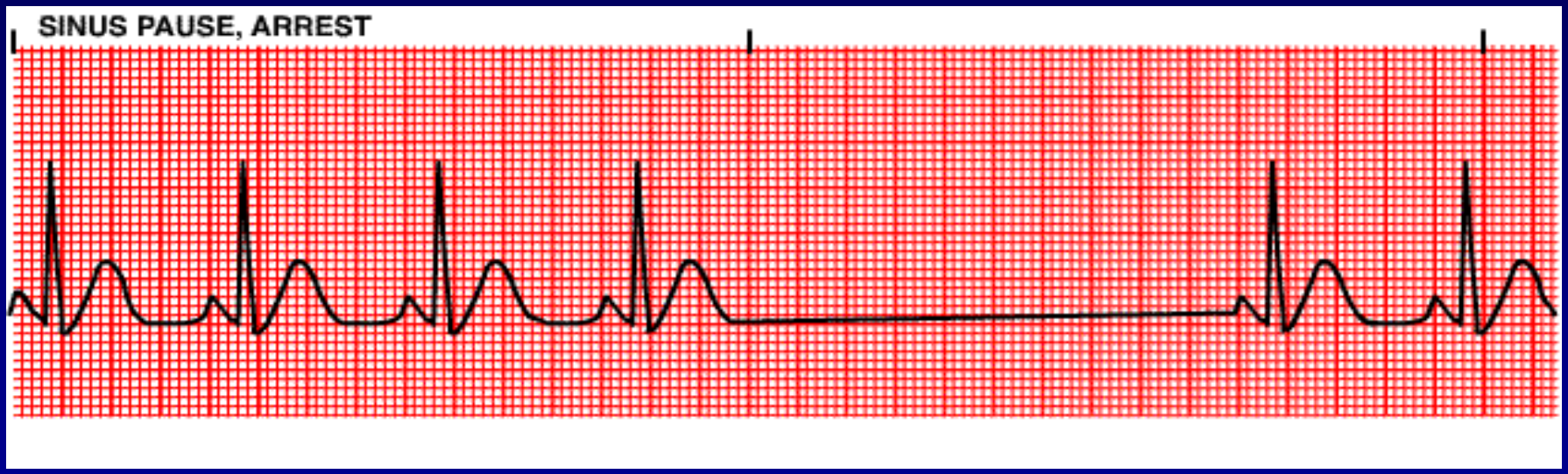
Sinus Bradycardia

- **Rate: less than 60** bpm
- **P wave:** sinus
- **QRS:** Normal (.06-.12)
- **Conduction:** P-R normal or slightly prolonged at slower rates
- **Rhythm:** regular
- This rhythm is often seen as a normal variation **in athletes, during sleep**, or in response to a vagal maneuver.
- **Pathological causes :** MI, SSS, hypothermia, hypothyroids, Increased ICP and drugs e.g. CCB, BB, digoxin.
- Treatment includes:
  - treat the underlying cause,
  - atropine,
  - isuprel, or
  - Patient with recurrent and persistence symptomatic bradycardia should be considered for pacemaker implantation .


SINUS TACHYCARDIA

Sinus Tachycardia

- **Rate: more than 100 bpm**
- **P wave:** sinus
- **QRS:** normal
- **Conduction:** normal
- **Rhythm:** regular
- The clinical significance of this dysrhythmia depends on the underlying cause. It may be normal.
- **Underlying causes include**:
  - Increased circulating catecholamines
  - CHF
  - Hypoxia
  - PE
  - Increased temperature
  - Stress
  - Response to pain
  - Anemia
  - or drugs
- Treatment includes identification of the underlying cause and correction.


SINUS PAUSE, ARREST

Sick sinus syndrome

Sick sinus syndrome is a cardiac conduction disorder where the hearts spontaneous pacemaker, does not send its electrical impulses quick enough to keep the heart beating as fast as the body requires.

Causes:

- CAD.
- SAN degeneration .
- connective tissue disease .
- metabolic disease .
- trauma .
- congenital disease.

Sinus Pause, Arrest

- **Rate:** normal with long pause
- **P wave:** those that are present are normal
- **QRS:** normal
- **Conduction:** normal
- **Rhythm:** The basic rhythm is regular. The length of the pause is not a multiple of the sinus interval.
- This may occur in individuals with healthy hearts. It may also occur with increased vagal tone, myocarditis, MI, and digitalis toxicity.
- If the pause is prolonged, escape beats may occur.
- The treatment of this dysrhythmia depends on the underlying cause.
  - If the cause is due to increased vagal tone and the patient is symptomatic, atropine may be indicated.

What Is Sick Sinus Syndrome?

When the sinus node malfunctions several different abnormalities may result:

(1) Inappropriate bradycardia.

(The heartbeat may become too slow for the demands of the body).

(2) Inappropriate tachycardia.

(The heartbeat may become too fast even at rest).

(3) Bradycardia-Tachycardia syndrome

(The heart rate may alternate between fast and slow).

(4) sinus pause or sinus arrest

There may be **sudden pauses** in the normal activity of sinus node of longer than 2 or 3 seconds' duration.

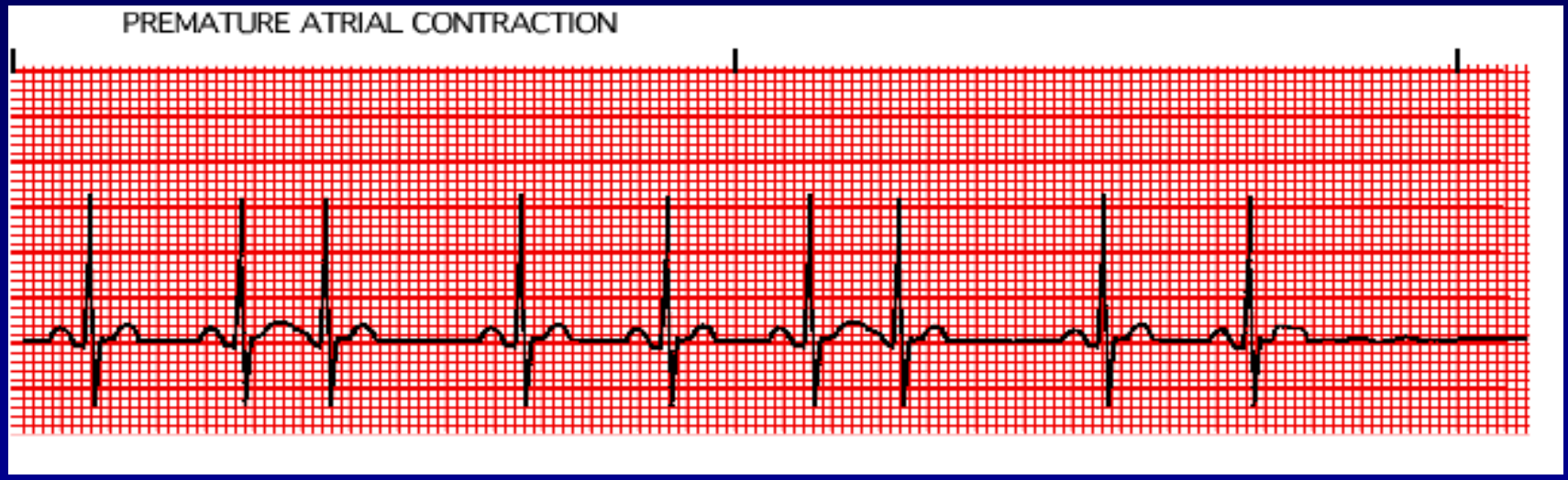
Sick Sinus Syndrome (SSS)

Therapy:

1. **Treat the etiology**
2. **Treat with drugs: anti-bradycardia agents, the effect of drug therapy is not good.**
3. **Artificial cardiac pacing .**

2-Atrial arrhythmia :

- Atrial ectopic beats
- Multifocal Atrial Tachycardia
- Paroxysmal Atrial tachycardia
- Atrial flutter
- Atrial fibrillation


PREMATURE ATRIAL CONTRACTION

Atrial Ectopic Beats

(Atrial extrasystoles, Premature Atrial Contraction PAC)

- Usually no symptoms but can give the sensation of a missed beat or abnormally strong beat .

ECG : -P wave -------- the preceding P wave has a different morphology

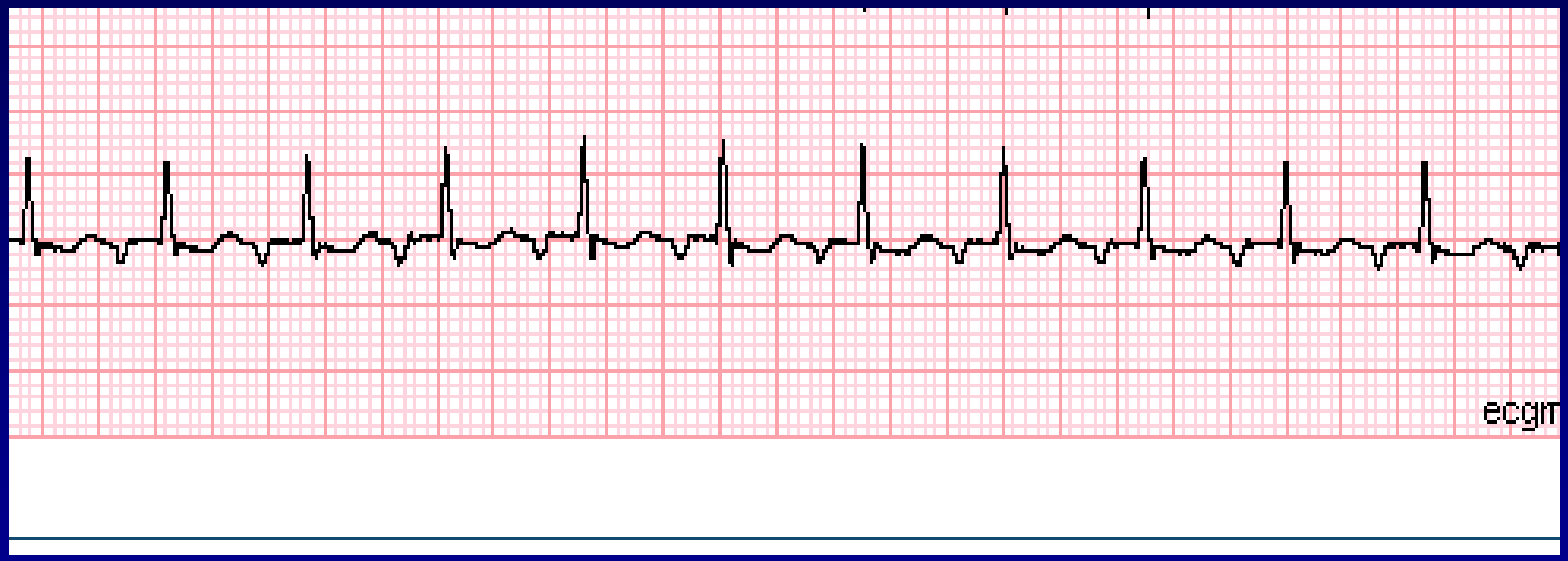
-QRS complex------ Occurs early in the cycle and normal morphology usually do not have compensatory pause .

- **Rate --**Normal

- PAC's occur normally in a non diseased heart and they can also result from CHF, ischemia and COPD
- However, if they occur frequently, they may lead to a more serious atrial dysrhythmia (atrial fibrillation)

- **Treatment : Rarely necessary ,BB can be used**

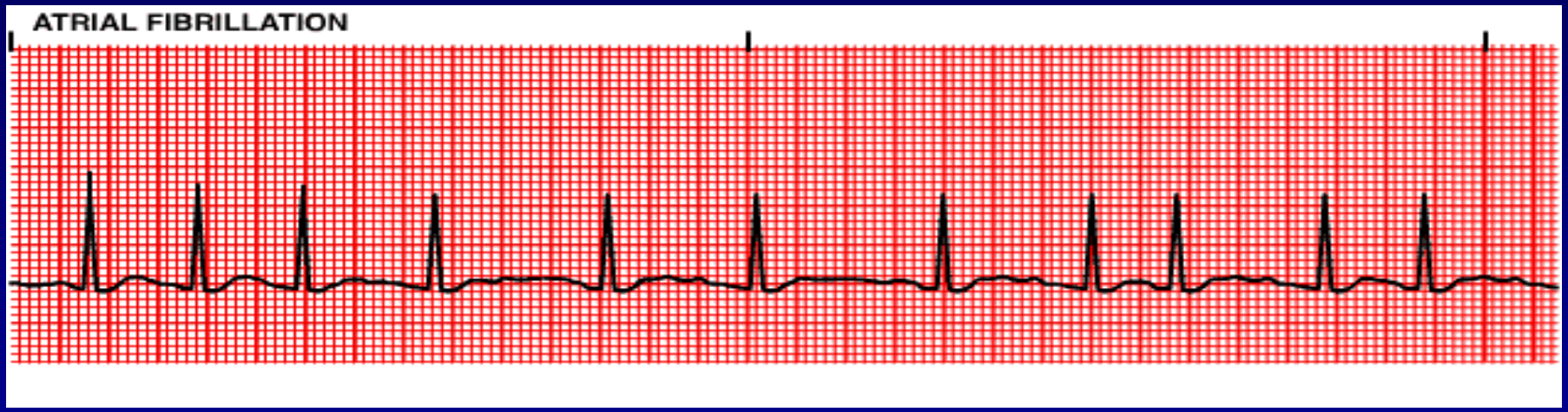
Atrial Tachycardia

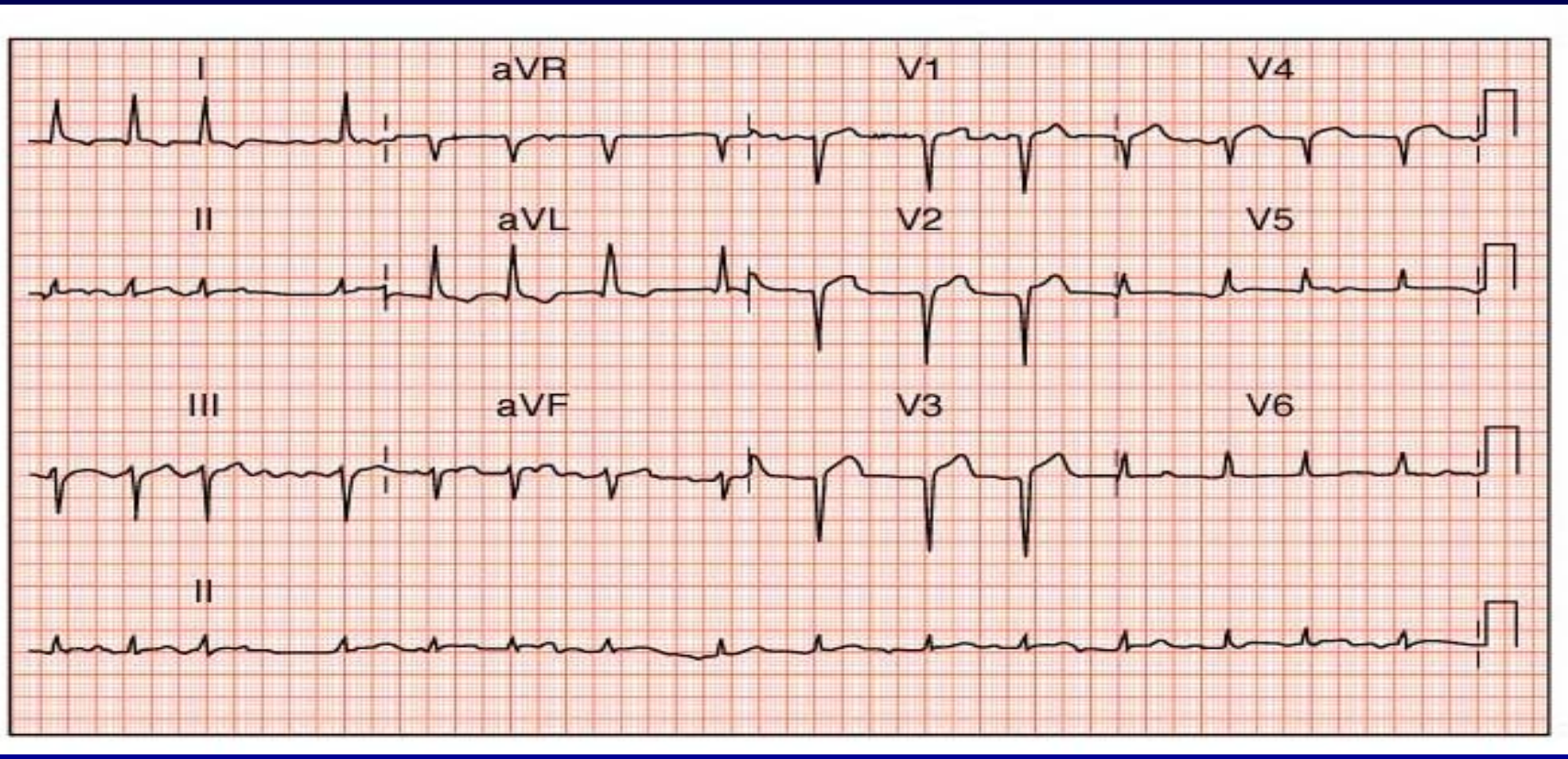


Atrial Tachycardia

Three or more consecutive ectopic atrial beats (non sinus P waves)

- **ECG - P wave:** Abnormal P wave morphology . P wave may precede, be buried in (sometimes not visualized), or immediately follow the QRS complex
  - **QRS:** normal (unless associated with aberrant ventricular conduction).
  - **Rate:** atrial 100-240/min: may conduct to ventricles 1:1, or 2:1, 3:1, 4:1 into the presence of a block.
- Occur with increased atrial automaticity ,Sinoatrial disease and digoxin toxicity
- This rhythm is often transient and non sustained and doesn't require treatment.
  - **AV blocking drugs** to control rapid ventricular response e.g. BB,CCB.
  - **EPS** and Catheter Ablation .


ATRIAL FIBRILLATION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Atrial Fibrillation

- The most common sustained cardiac arrhythmia
- The prevalence increased by age affecting those aged 60 to 64 years 1 %.and occur 9% of those aged over 80 years
- The mechanism is abnormal automaticity and Re-entry

ECG in Atrial Fibrillation Ch Ch by :

- **Rhythm: irregularly irregular.**
  (This is the hallmark of this dysrhythmia).
- **P wave:** not present; wavy baseline is seen instead.
- **Rate:** atrial rate usually between 400-650/bpm.
- **Conduction:** variable AV conduction; if untreated the ventricular response is usually rapid.

Classification of atrial fibrillation

1- Paroxysmal A.Fib : Intermittent episodes which self-terminate within7 days .

2- Persistent A.Fib : Prolonged episodes that can be terminated by electrical or chemical cardioversion .

3-Perminanet A;Fib .

Atrial Fibrillation

Common causes of atrial fibrillation	
CAD	Pericardial
Valvular heart disease	Hyperthyroidism disease
Cardiomyopathy	Alcohol
Congenital heart disease	Chest infection
Hypertension	Idiopathic (lone atrial fibrillation)
Pulmonary embolism	

Management of atrial fibrillation

The Main objectives for management :

1- Rhythm control

-Chemical cardioversion

-Flecainid and propafenone ,dronedarone and amiodarone

- Electrical cardioversion

- Catheter ablation (Pulmonary vein isolation)

2- Rate control : AV nodal blocking agent

-BB,CCB and digoxin

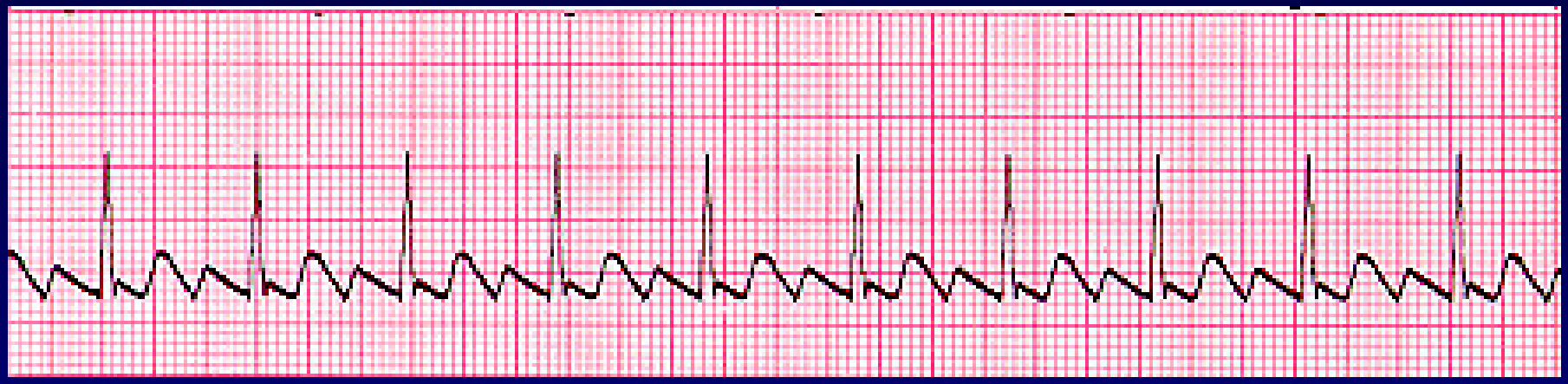
3- Prevention of thromboembolism

According CHA2DS2VASc

-Warfarin

- New oral anticoagulant For Non valvular atrial fibrillation

Dabigatran ,Rivroxxban ,Apixaban



ATRIAL FLUTTER

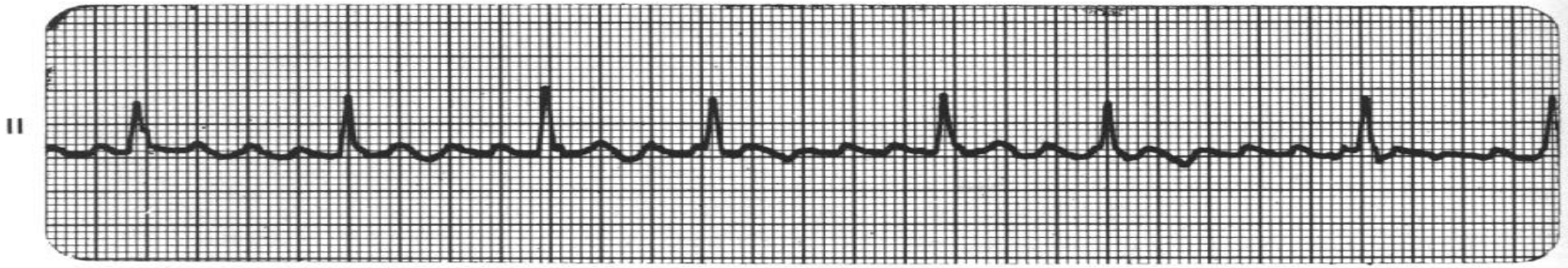
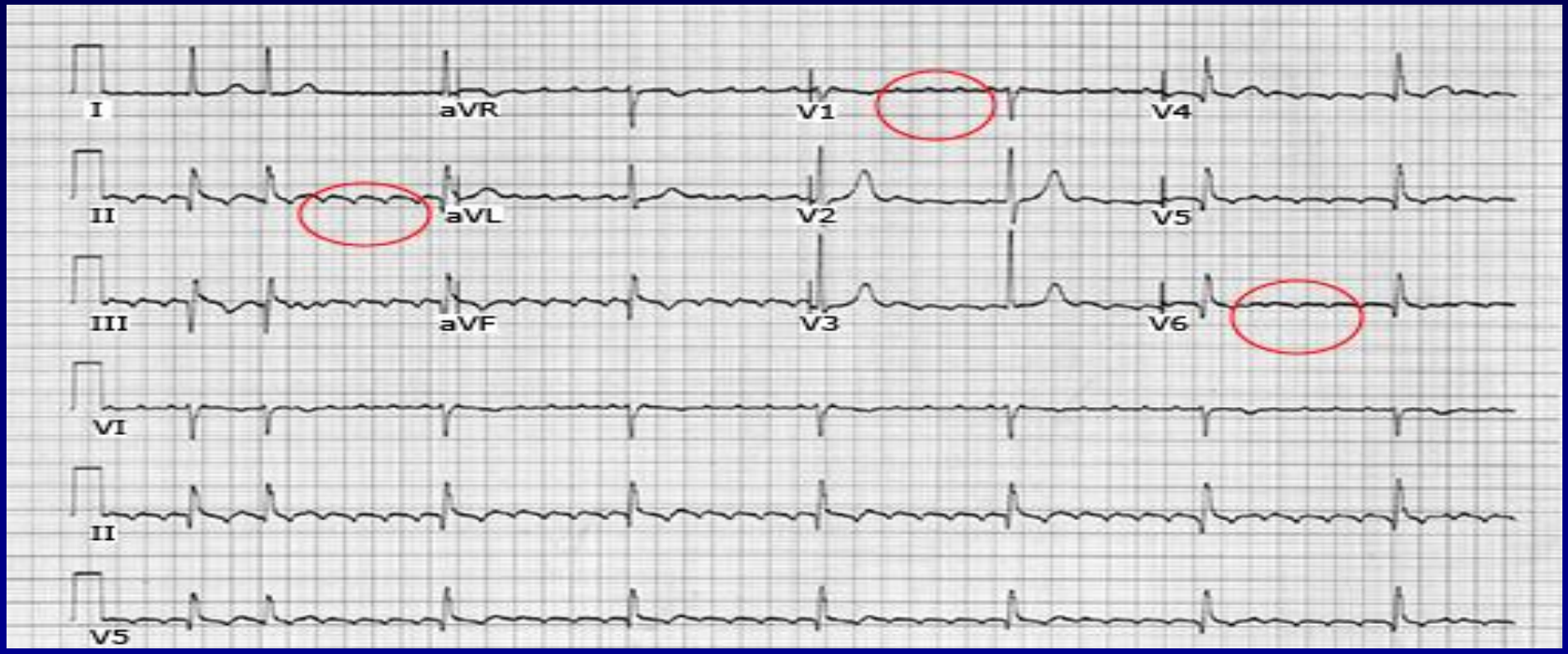


Fig. 13-3. Note the variable ventricular rate in this patient with atrial flutter.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VI
II
V5

Atrial Flutter

✓Atrial Flutter is less common than fibrillation
✓Most AF have a reentry loop in right atrial

ECG in Atrial Flutter Ch Ch by :

P wave : Rapid regular atrial undulations (flutter or "F" waves or "sawtooth" pattern)

Rate : 240-340 per minute

QRS : May be normal or wide .Rate and regularity of QRS complexes depend on the AV conduction sequence

AV conduction : Ratio of flutter waves to QRS complexes is usually fixed and an even number (e.g.,2:1, 4:1), but may vary.

Causes of atrial flutter

- COPD
- Rheumatic Heart disease **(mitral or tricuspid valve disease),**
- Coronary artery disease
- Heart failure
- Atrial septal defect
- Post cardiac surgery for congenital heart disease
- hypertension
- hyperthyroidism

Symptoms:

Depend on underlying disease and ventricular rate

With rapid ventricular rate:

-Palpitation -weakness

-Syncope,. -Dizziness

-Shortness of breath,

-May develop angina and CHF.

Management of Atrial Flutter

The Main objectives for management :

1- Rhythm control

- Chemical cardioversion (antiarrhythmics drugs)

-Ibutilide , Defetilide

- Electrical cardioversion (good response better than A.fib)

- Catheter ablation (is highly successful for chronic A. Flutter)

2- Rate control : AV nodal blocking agent

-BB,CCB,Digoxin

3- Prevention of thromboembolism

-Warfarin

- New oral anticoagulant

Dabigatran ,Rivroxxban ,Apixaban

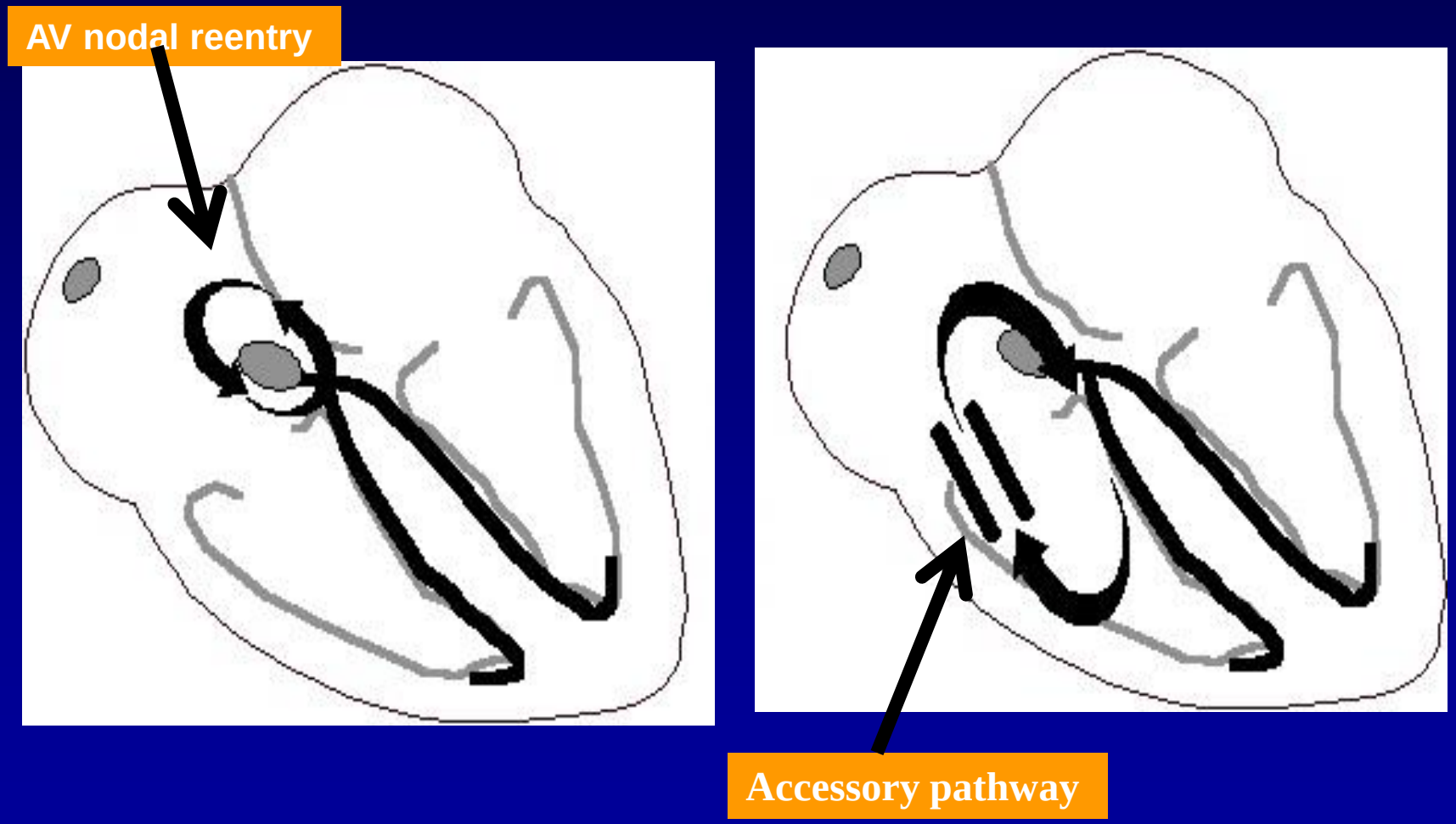
3-Junctional arrhythmia

- Premature Junction Contraction
- Junction Tachycardia
- Junction Escape Rhythm
- paroxysmal supraventricular tachycardia
  - AVNRT
  - AVRT
  - WPW

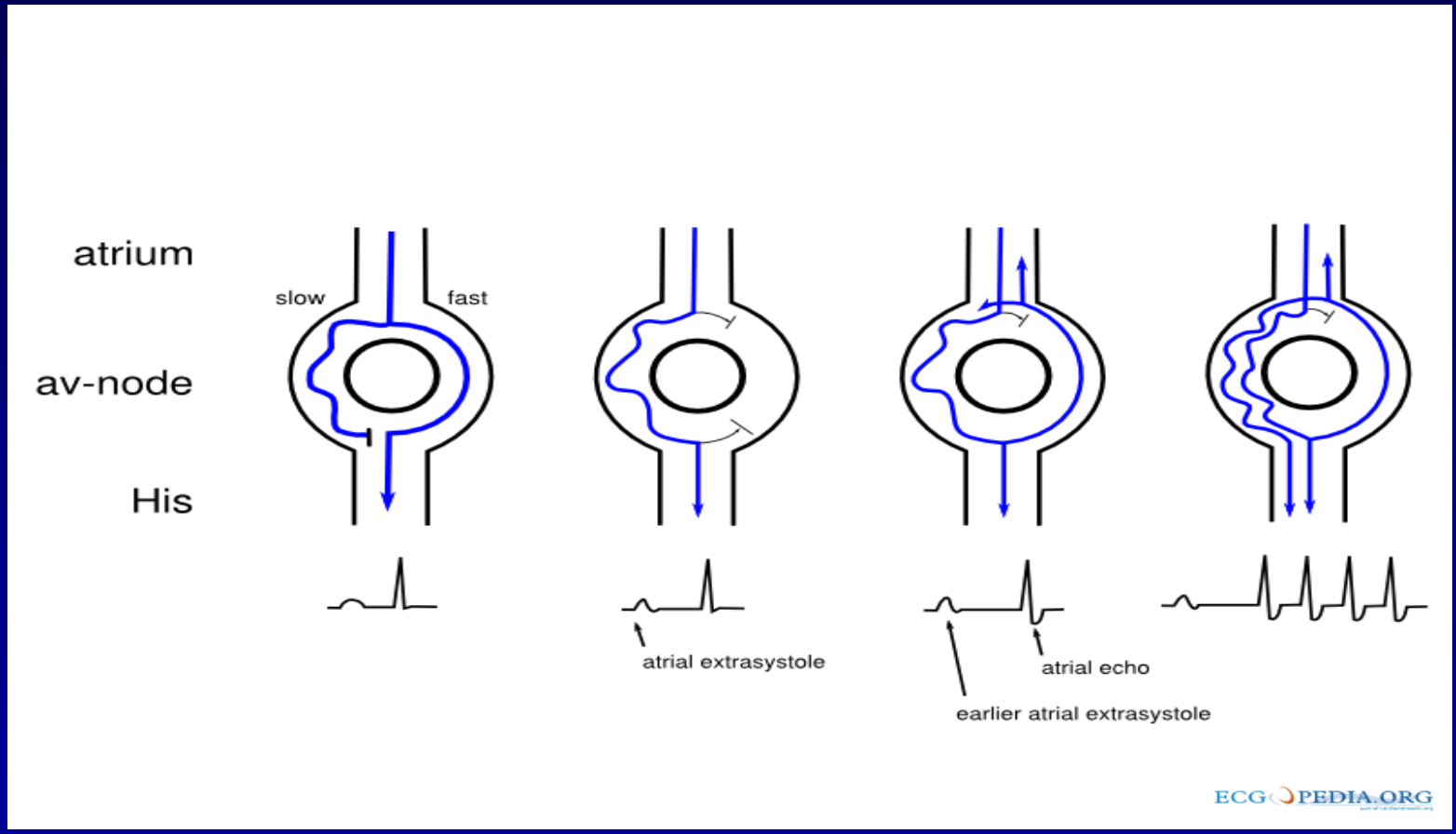
Paroxysmal Supraventricular Tachycardia

- Most PSVT is due to reentrant mechanism.
- The incidence of PSVT is higher in AVNRT (atrioventricular node reentry tachycardia) and less in AVRT (atioventricular reentry tachycardia).
- Occur in any age individuals, usually no structure heart disease.

Reentrant Mechanism



AV nodal Reentry



Paroxysmal Supraventricular Tachycardia

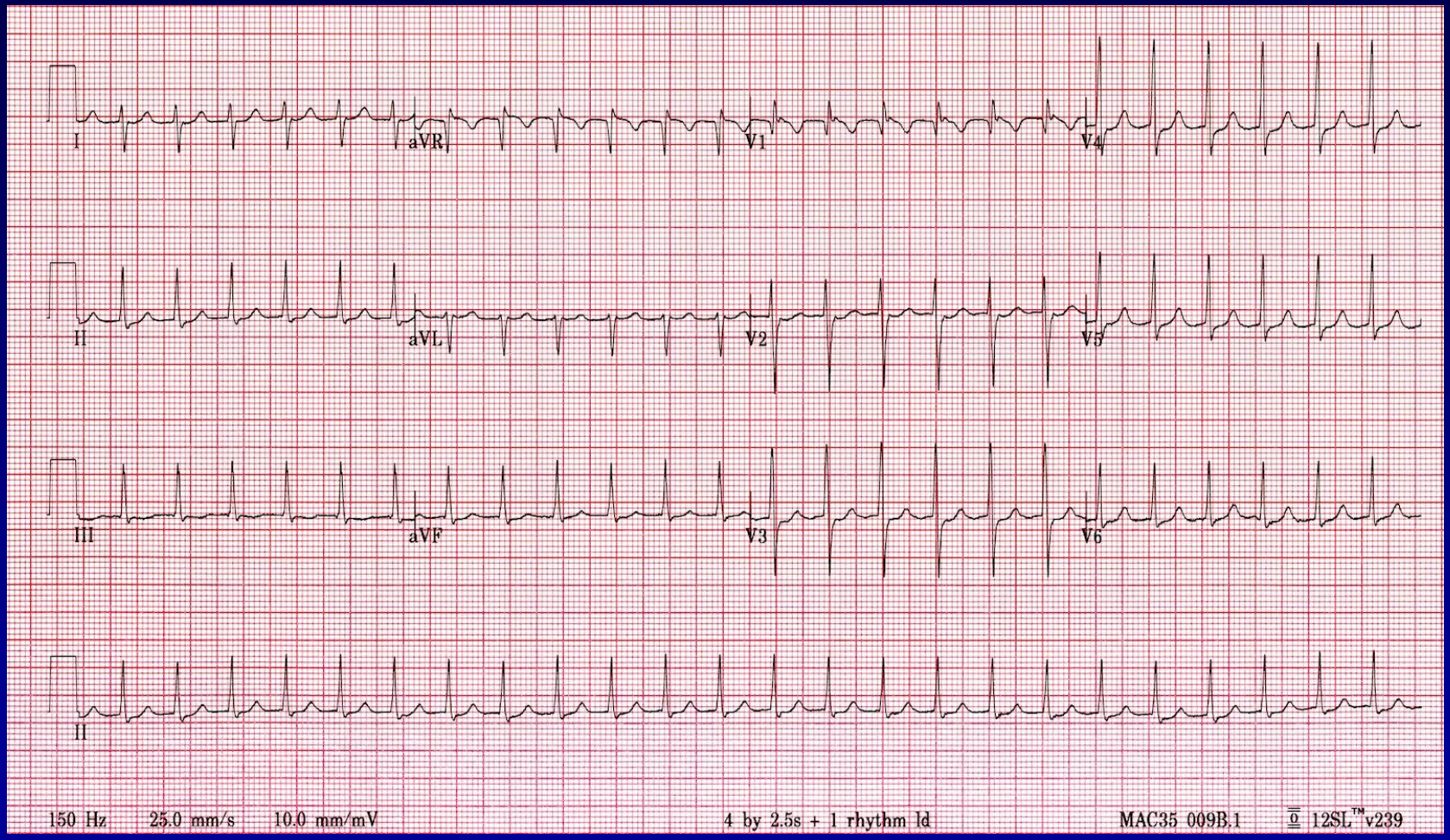
Manifestation:

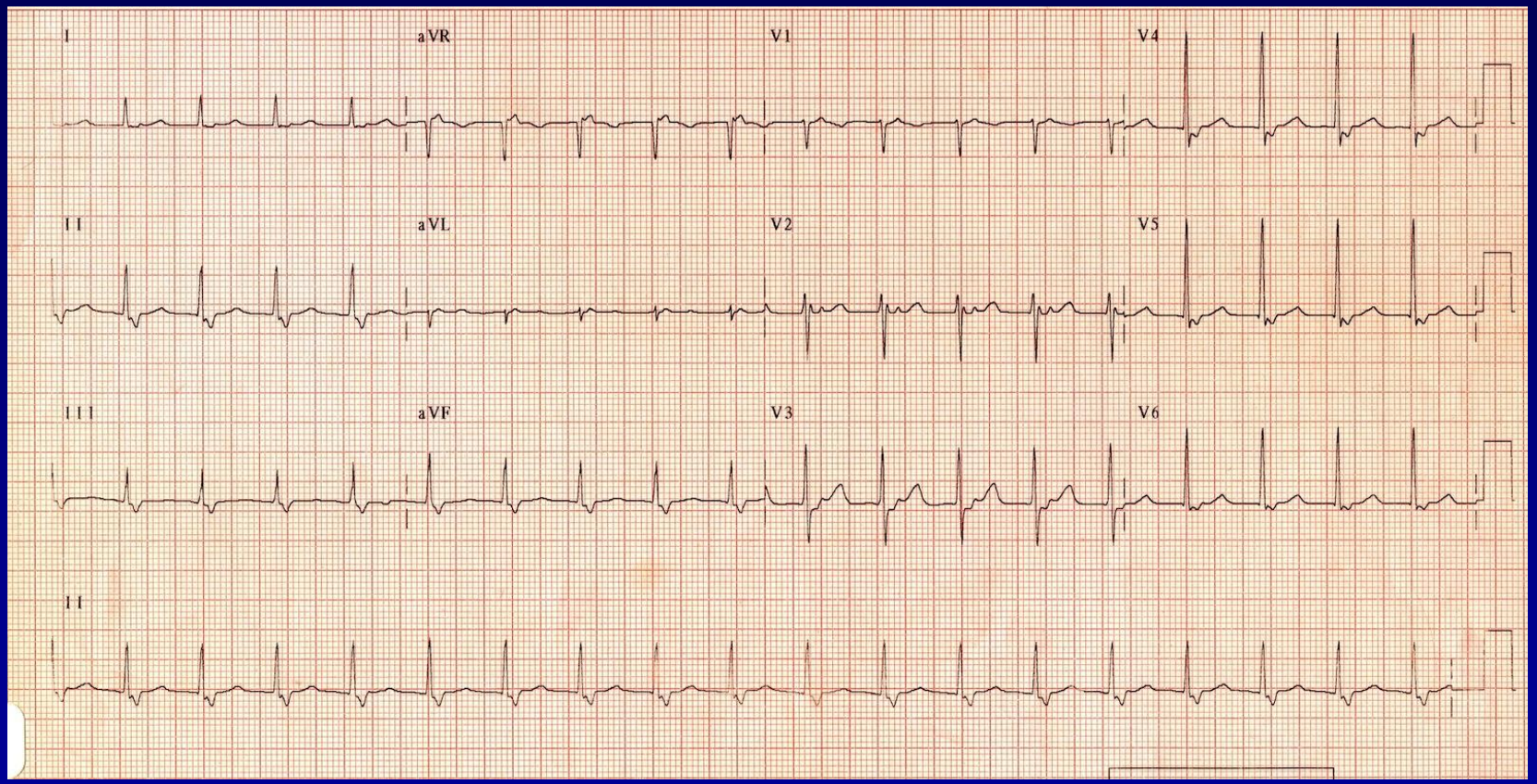
- Occur and terminal abruptly.
- Palpitation, dizziness, syncope, angina, heart failure and shock.
- The sever degree of the symptom is related to ventricular rate, persistent duration and underlying disease

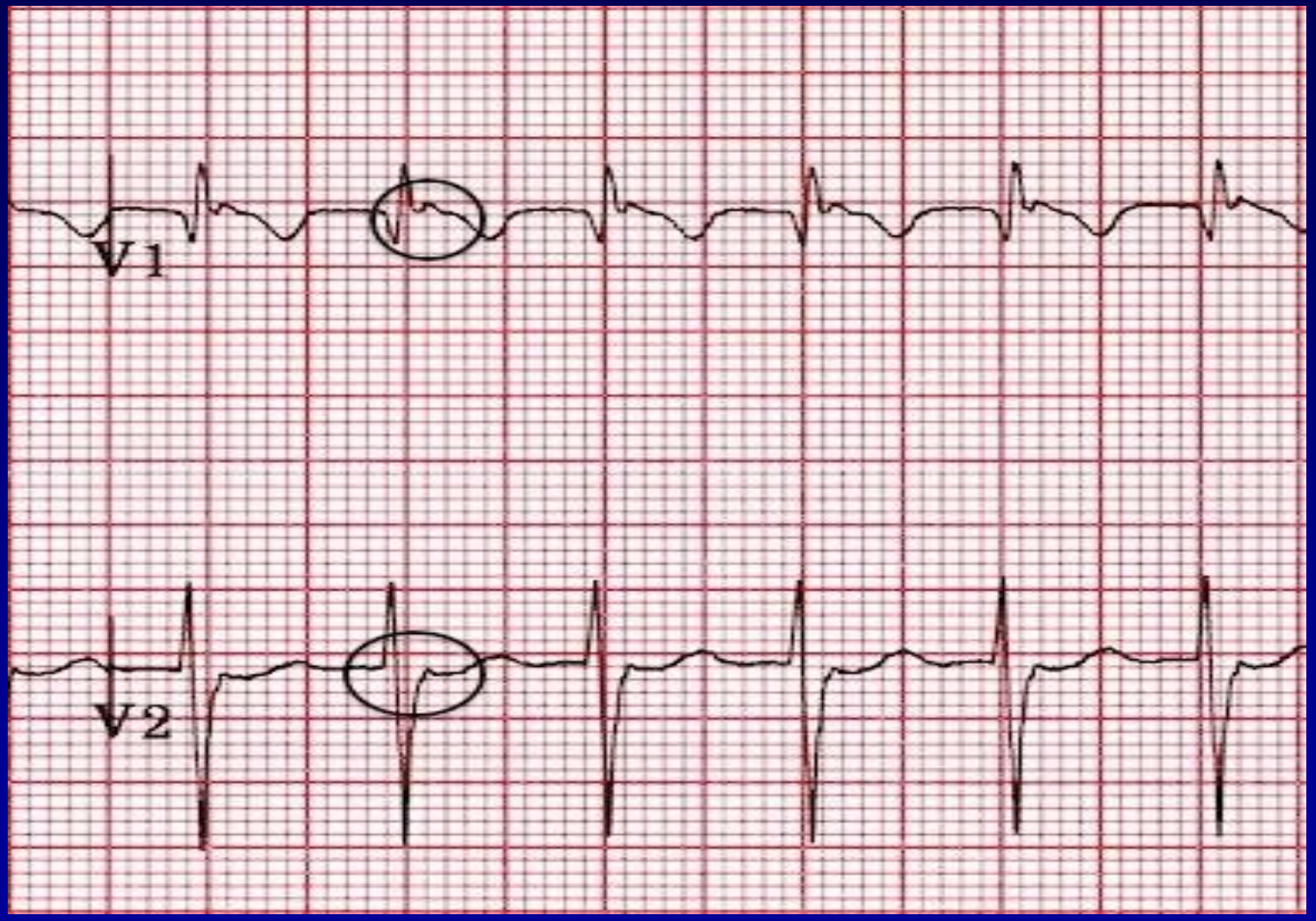
Paroxysmal Supraventricular Tachycardia

ECG characteristic of AVNRT

1. Heart rate is 150-250 bpm, regular
2. QRS complex is often normal, wide QRS complex is with aberrant conduction
3. Negative P wave in II III aVF, buried into or following by the QRS complex.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
150 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm ld
MAC35 009B.1
12SL™v239


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


V1
V2

Paroxysmal Supraventricular Tachycardia

Therapy:

AVNRT & orthodromic AVRT

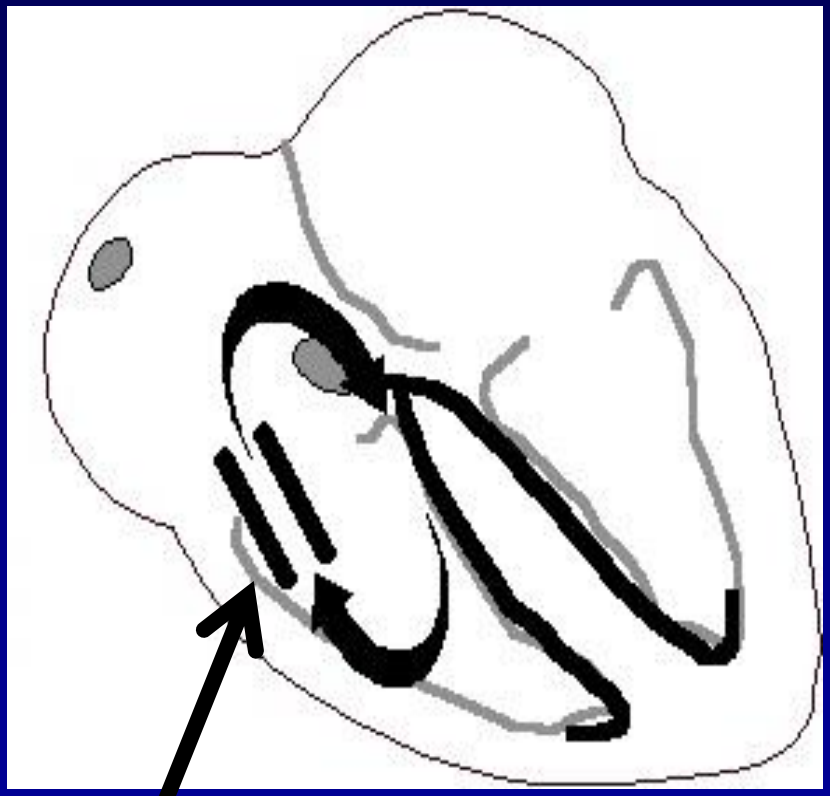
(Narrow complex SVT)

1. Increase vagal tone: carotid sinus massage, Valsalva maneuver.if no successful,
2. Drug: -Adrenosine, Verapamil, BB
   -Antiarrhythmia classIc or class III
3. DC shock (If patient hemodynamic unstable)
4. Catheter ablation .

Pre-excitation syndrome (W-P-W syndrome)

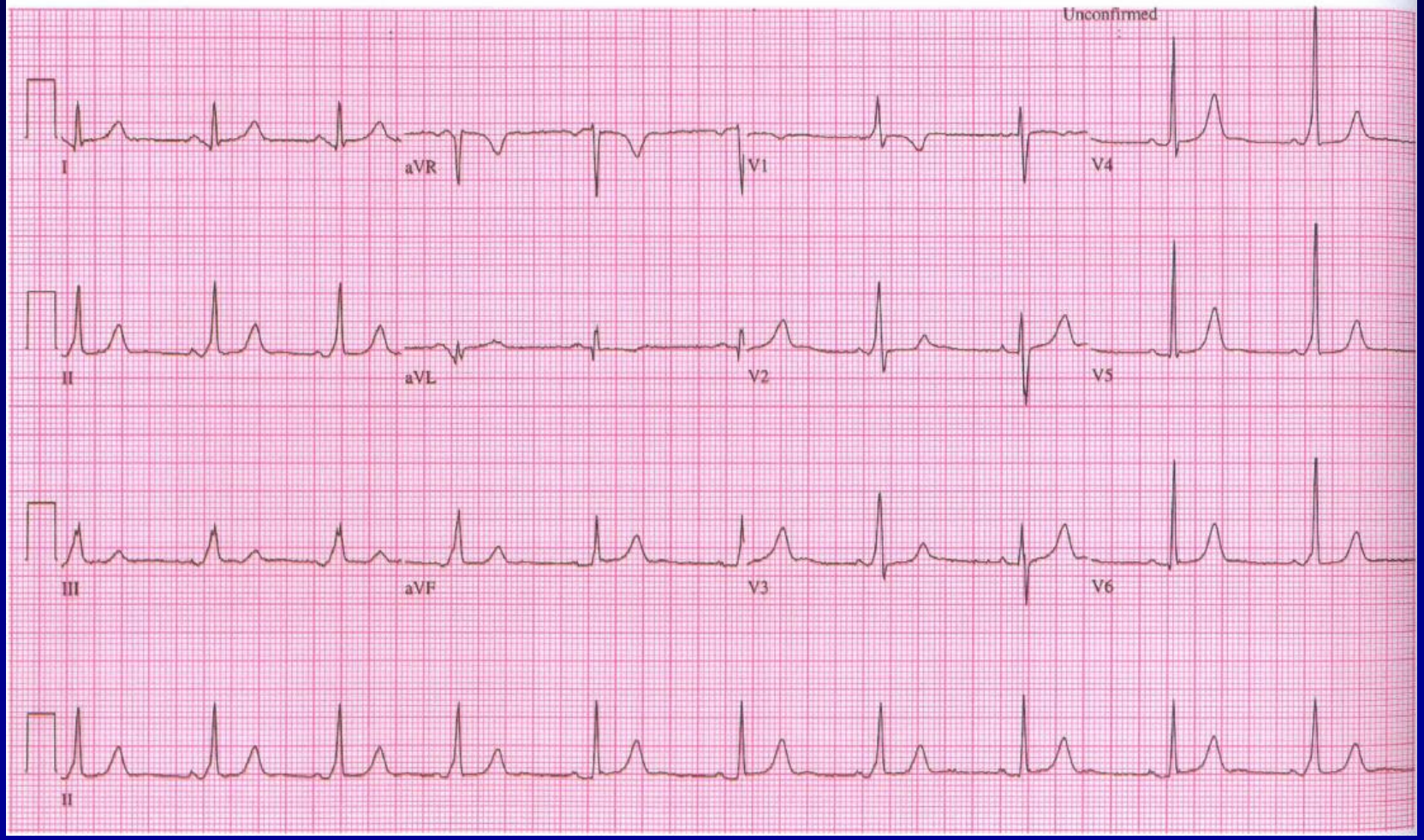
- Accessory pathway or bypass tract between atrium and the ventricle (kent bundles)
- This often produces a short interval with a delta wave (preexcitation) at the onset of wide, slurred QRS complex.
- Usually no structure heart disease, occur in any age individual.
- Two types Orthodromic and antidromoic AVRT.

Accessory Pathway

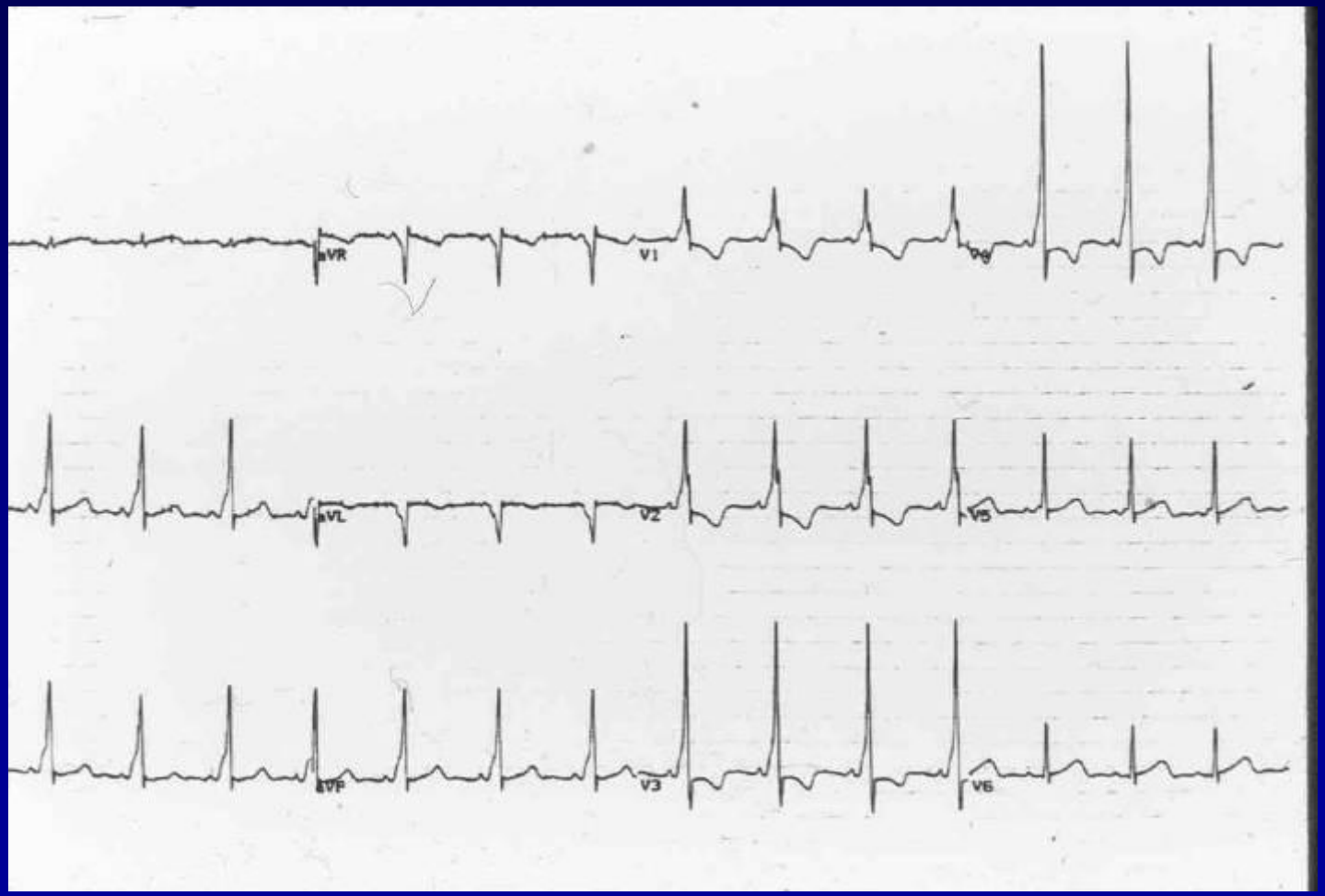


TRACING 36

24-year-old male seen in the office complaining of intermittent brief periods of rapid heart action



WPW



WPW syndrome

Manifestation:

- Occur in 0.1 -0.3 of the population
- Palpitation, syncope, dizziness
- Arrhythmia: 80% tachycardia is AVRT, 15-30% is AFi, 5% is AF,
- May induce ventricular fibrillation

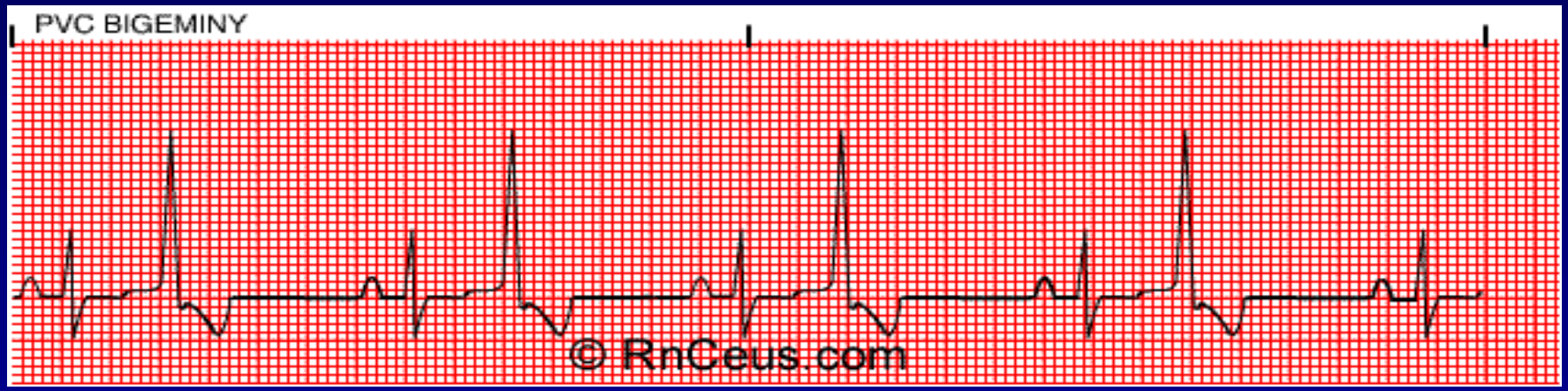
WPW syndrome

Therapy:

- **Incidental WPW** without symptoms don't require specific therapy.
- **DC shock:** WPW with SVT, AF or Afi produce angina, syncope and hypotension
- If hemodynamic stable : **antiarrhythmia** Class 1a,class 1c and class III
- **Do not used AV nodal blocking agent** BB,CCB and digoxin
- **Ablation :** RFA has become the procedure of choice.

4.Ventricular arrhythmia

- Premature Ventricular Contraction
- Ventricular Tachycardia
- Ventricular fibrillation


PVC BIGEMINY
© RnCeus.com

Ventricular Ectopic Beats

PVC ,Extrasystoles

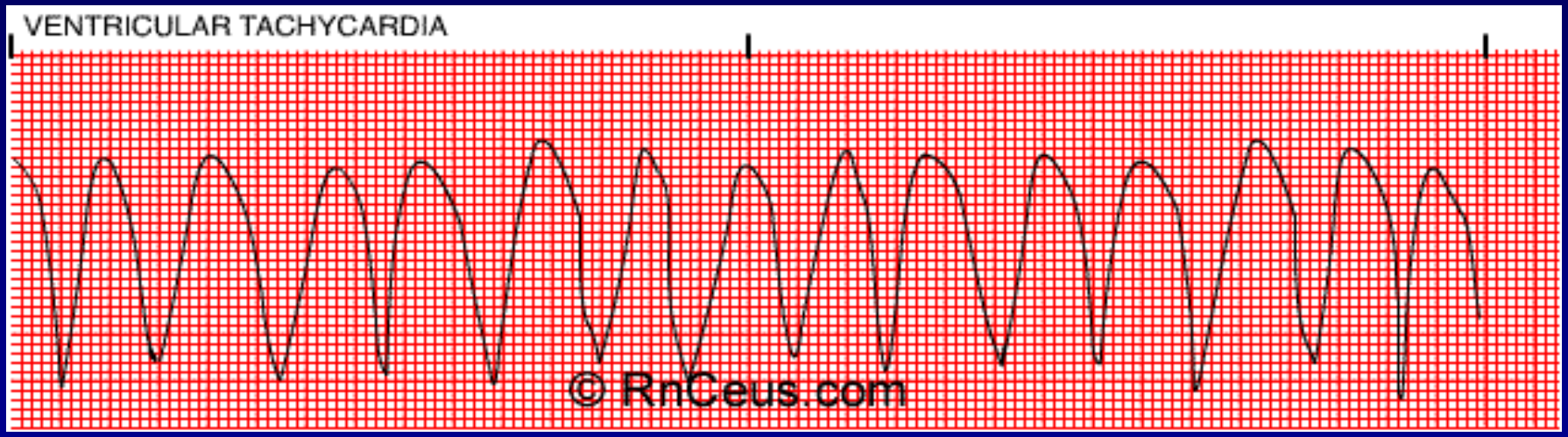
- **P wave: usually No Pwave**
- **QRS: wide > 0.12 seconds; morphology is bizarre**
- **Conduction:** the impulse originates below the branching portion of the Bundle of His; full compensatory pause is characteristic.
- **Rhythm:** irregular. PVC's may occur in singles, couplets or triplets; or in bigeminy, trigeminy or quadrigeminy.

- **PVCs can occur in healthy hearts:**

  If Symptomatic ------BB, and If significant Catheter Ablation ??
- **PVCs with diseased hearts Post MI or HF :**

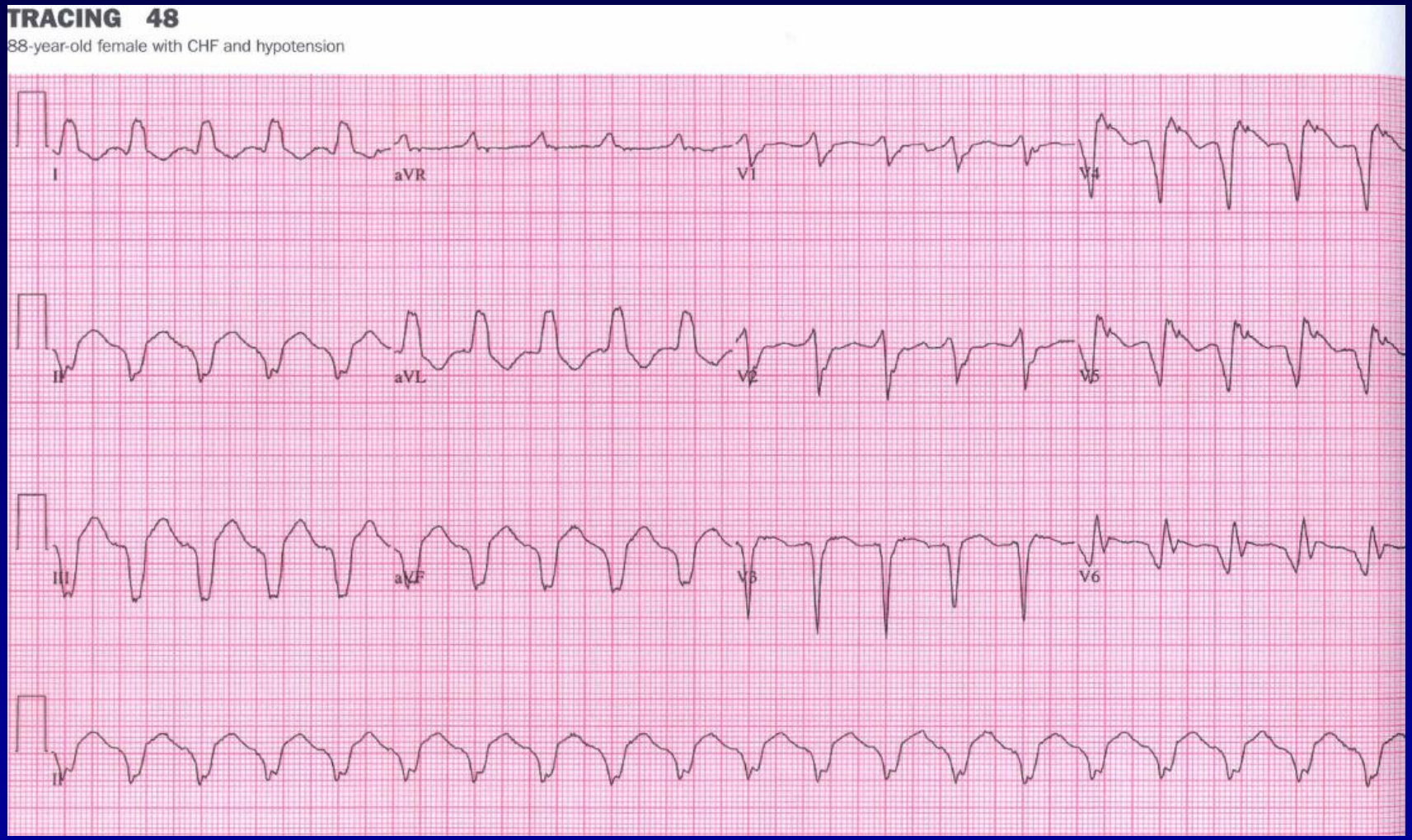
  Treatment : -B-blockers

  - If significant >20%------ ablation


VENTRICULAR TACHYCARDIA
© RnCeus.com

TRACING 48

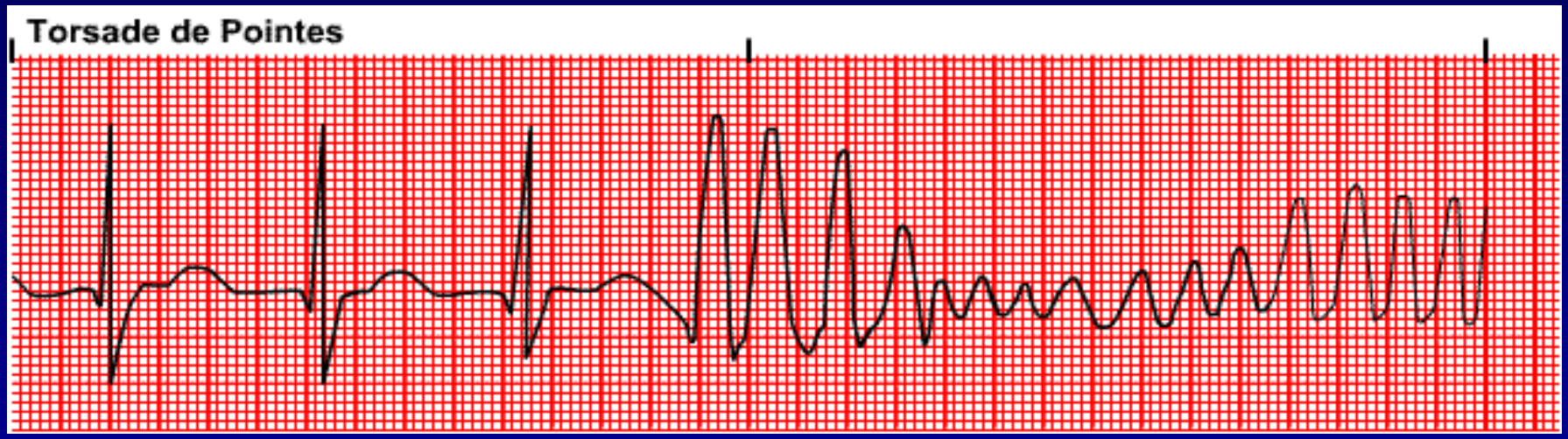
88-year-old female with CHF and hypotension



Ventricular Tachycardia

Ventricular tachycardia almost always occurs in diseased hearts.

- **Rate:** usually between 120to 220/bpm, but can be as rapid as 250/bpm
- **P wave:** obscured if present and are unrelated to the QRS complexes.
- **QRS:** wide and bizarre morphology
- **Rhythm:** three or more ventricular beats in a row; may be regular or irregular.
- **Some common causes are:**
  - Chronic CAD (ventricular aneurysms)
  - acute MI
  - CHF
  - digitalis toxicity
- **Symptoms :** dizziness ,dyspnea and syncope .
- **Management :**
  - Synchronized DC **cardioversion** for unstable patient .
  - **Amiodarone** for hemodynamic stable patient .
  - **B-blockers .**
  - **Correct** acidosis ,hypoxia and Hyperkalemia and hypomagnesaemia .
  - **ICD** may needs .


Torsade de Pointes

Torsade De Pointes (ventricular Tachycardia)

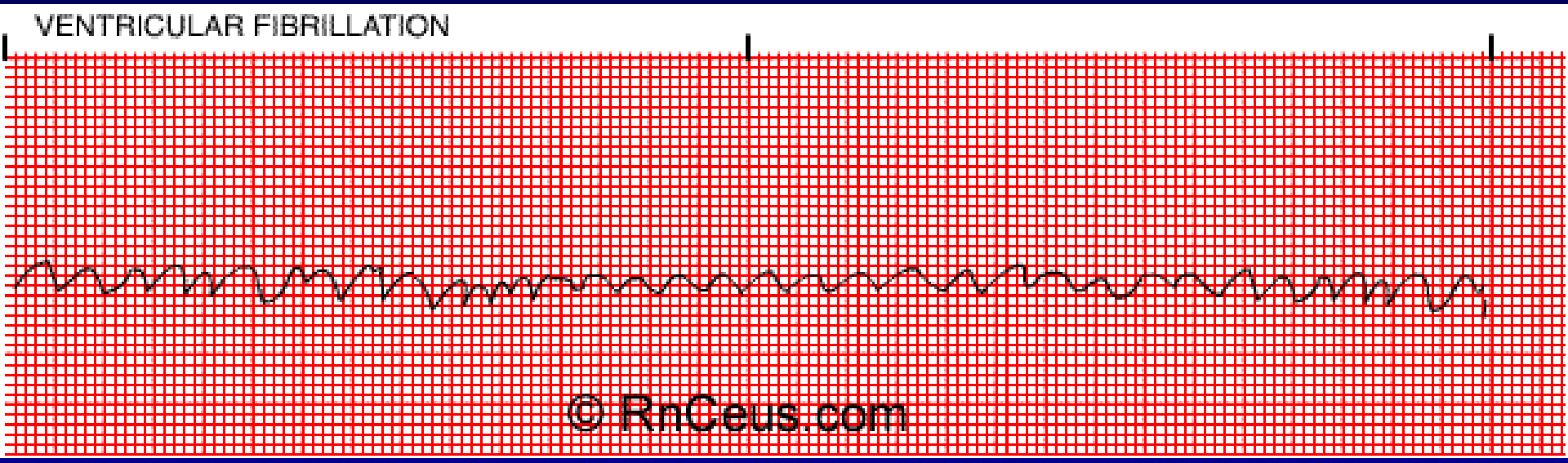
- This form of Polymorphic VT is a complication of prolonged QT interval

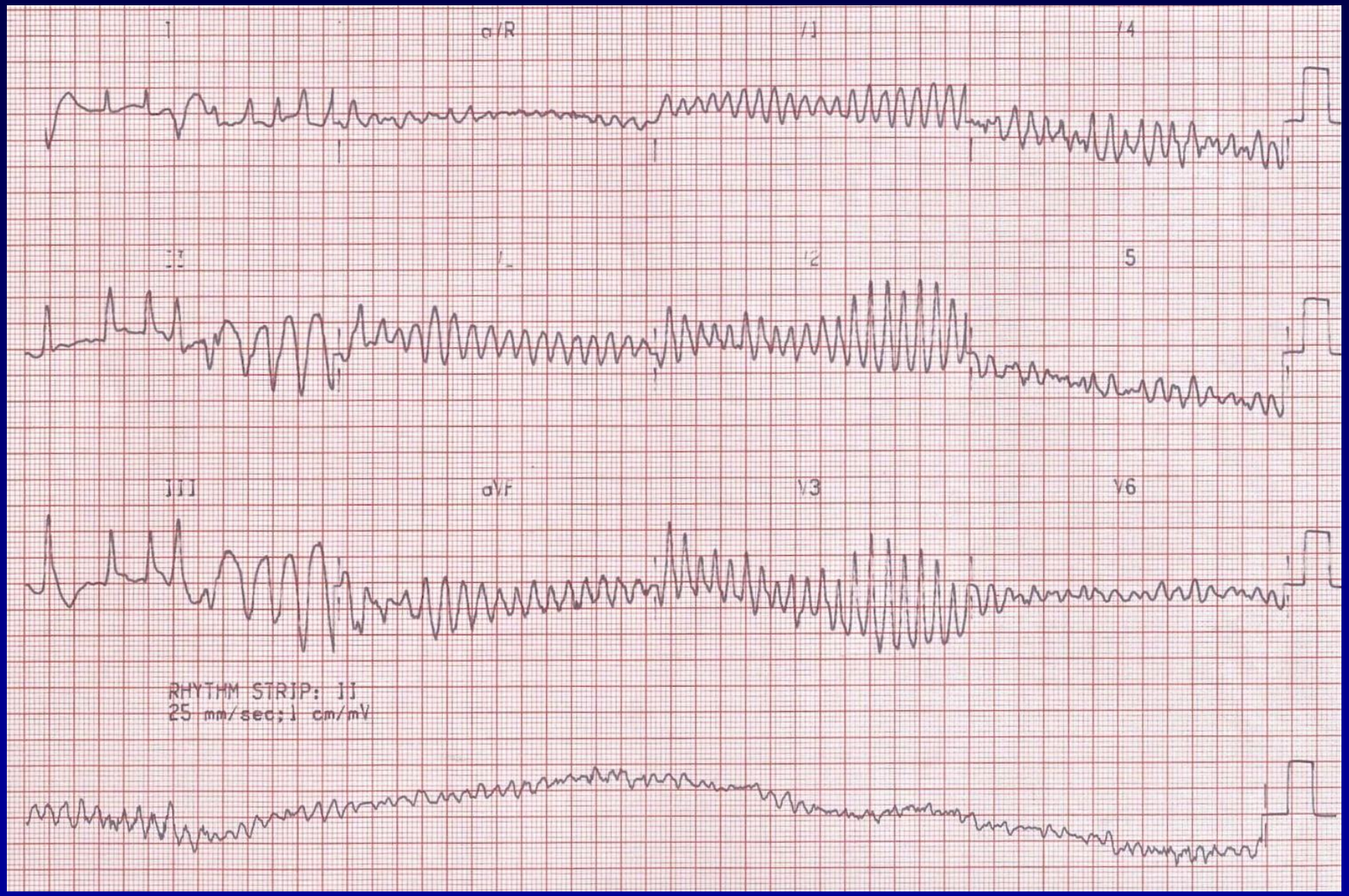
- **Paroxysmal** –Starting and stopping suddenly, usually non sustained
  But may degenerated to ventricular fibrillation .
- Hallmark of this rhythm is the upward and downward deflection of the QRS complexes around the baseline.
- The term **Torsade de Pointes** means **"twisting about the points."**

Caused by :

- Antiarrhythmia drugs which lengthen the QT interval such as quinidine
- electrolyte imbalances, particularly Hyperkalemia and hypomagnesaemia
- myocardial ischemia

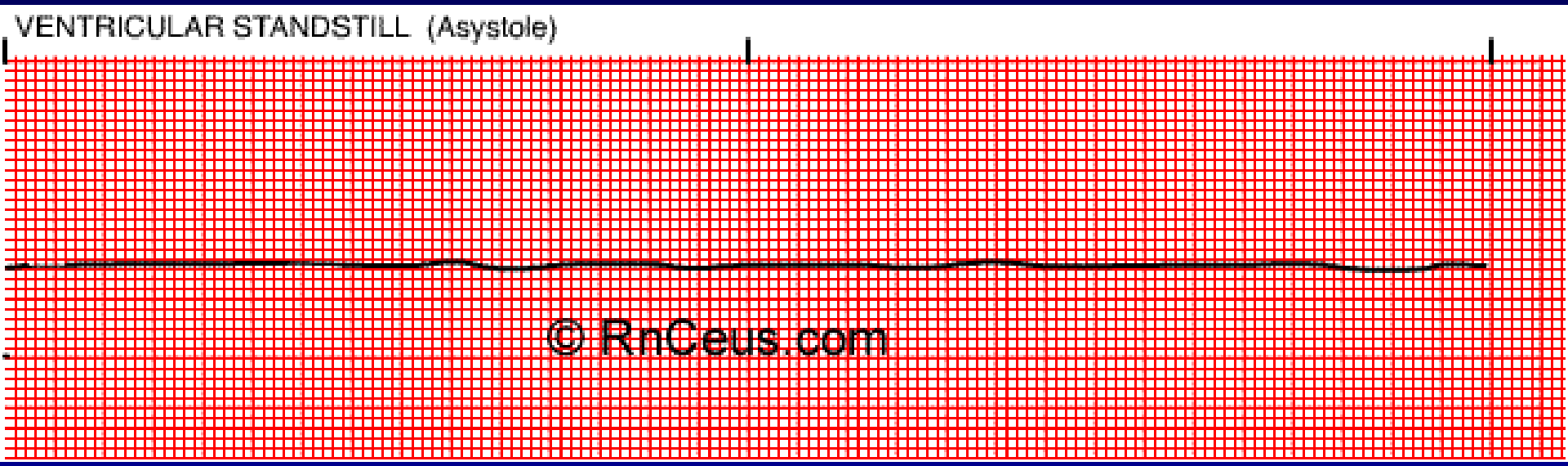
- **Treatment:**
  - Synchronized cardioversion is indicated when the patient is unstable.
  - IV magnesium
  - IV Potassium to correct an electrolyte imbalance
  - Overdrive pacing


VENTRICULAR FIBRILLATION
© RnCeus.com


I
aVR
V1
V4
II
V2
5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

Ventricular Fibrillation

- **The causative cause of sudden cardiac death**
- **Rate:** unattainable
- **P wave:** **No P wave ,**obscured by ventricular waves
- **QRS:** not apparent
- **Rhythm:** chaotic electrical activity
- This dysrhythmia results in the absence of cardiac output.
- Almost always occurs with serious heart disease, especially acute MI.
- **The course of treatment for ventricular fibrillation includes**:
  - immediate defibrillation and ACLS protocols.
  - Identification and treatment of the underlying cause is also needed.

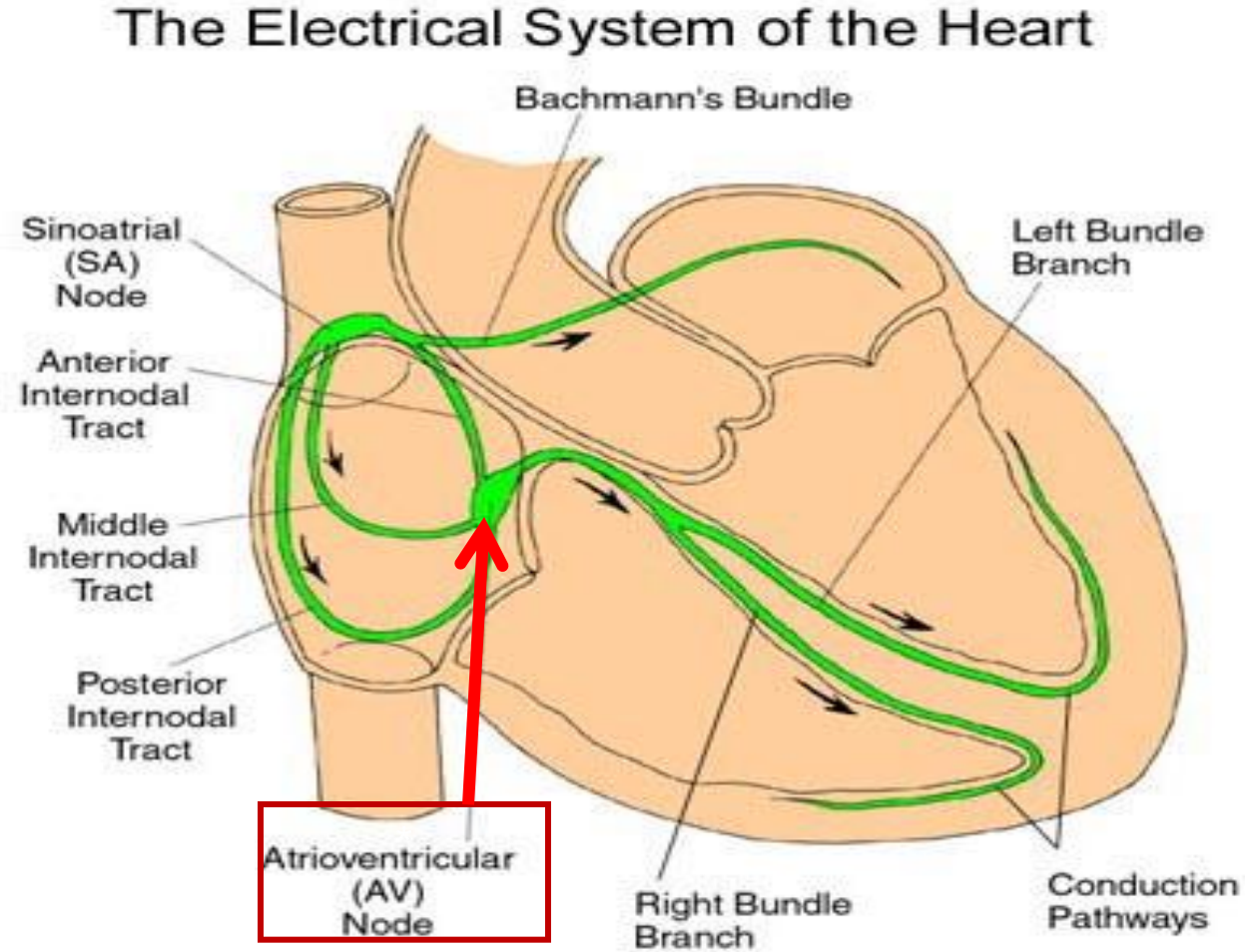

VENTRICULAR STANDSTILL (Asystole)
© RnCeus.com

Ventricular Standstill (ASYSTOLE)

- **Rate:** none
- **P wave:** may be seen, but there is no ventricular response
- **QRS:** none
- **Conduction:** none
- **Rhythm:** none
- Asystole occurs most commonly following the termination of atrial, AV junctional or ventricular tachycardias. **This pause is usually insignificant.**
- Asystole of longer duration in the presence of acute MI and CAD is frequently **fatal.**
- **Interventions include:**
  - CPR,
  - artificial pacing, and
  - atropine.

1. **Abnormal Cardiac dconduction**
    1. Atrio-ventricular block
    2. Intra-ventricular block

THE CONDUCTION SYSTEM OF THE HEART



AV Nodal Blocks (heart blocks)

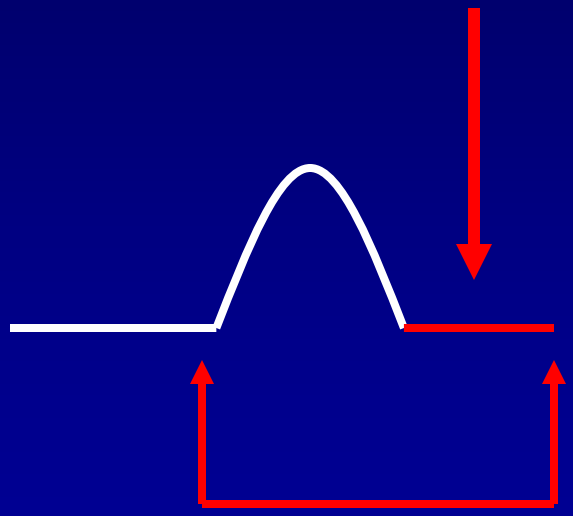
- **Disturbances of the conduction** through the heart, occurring **at the AV Node**
- AV Node – damaged/diseased – delay or total block of impulses at the AV Node
- This conduction defect can be seen on the ECG

Causes

- Increased vagal tone (parasympathetic nervous system)
- IHD (MI)
- Endocarditis
- Degeneration (age)
- Sclerosis (Aortic)
- Cardiac surgery trauma

AV Node

- AV nodal conduction time is represented on the ECG as the PR segment.



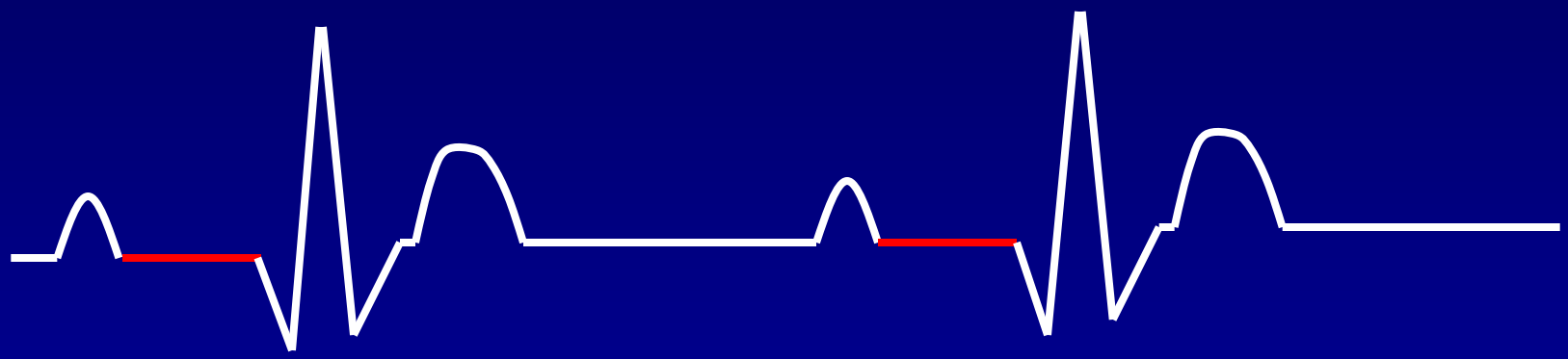
- But - we always measure the PR interval.

First Degree Heart Block (1º)

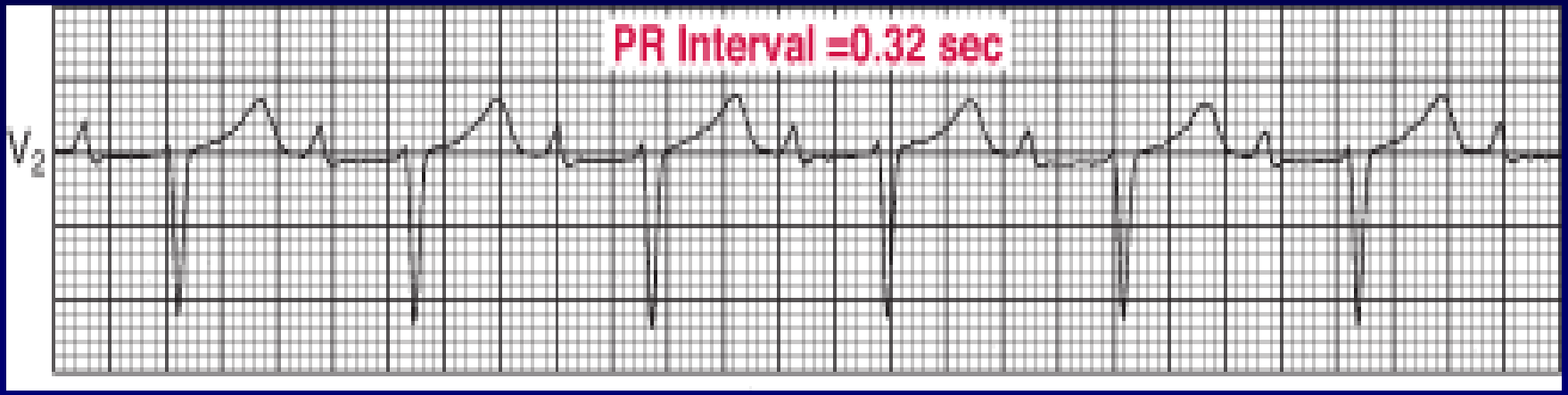
- SA Node – normal
  - Normal P wave
- AV Node conducts more slowly than normal
  - **Prolonged PR Interval**
- Rest of conduction is normal
  - Normal QRS

First Degree Heart Block (1º)

- PR Interval > 0.2 seconds (5 small sq)



- Note – the PR Interval is constant


PR Interval =0.32 sec
V2

Second Degree Heart Block (2°)

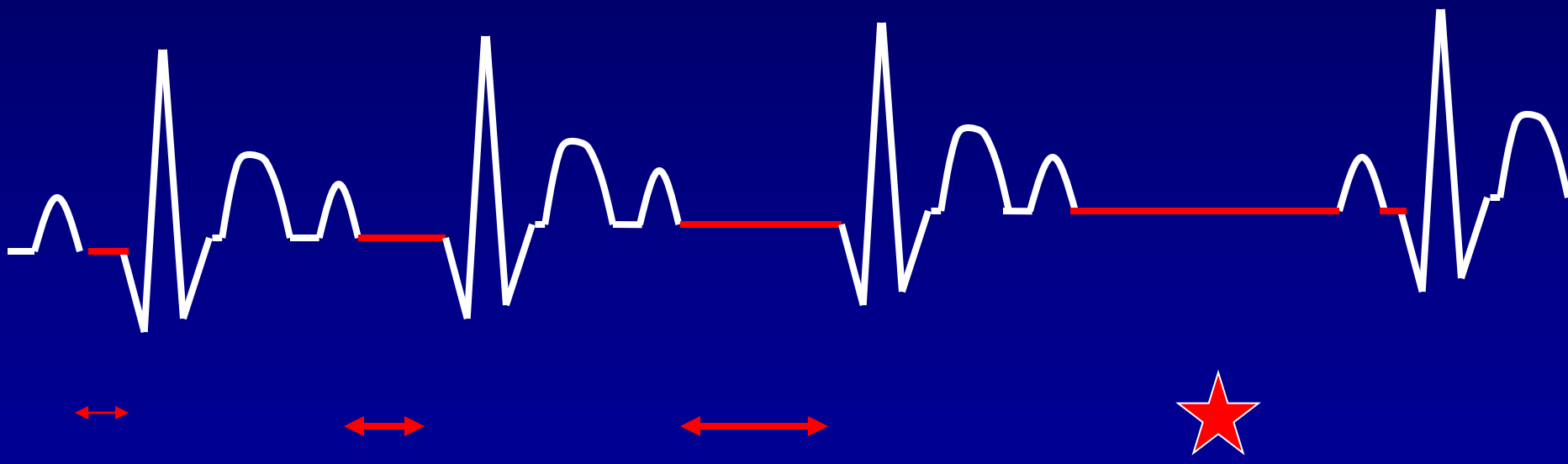
- Mobitz Type I (Wenkebach)
- Mobitz Type II

Second Degree Heart Block (2º)
Mobitz Type I
(Wenkebach)

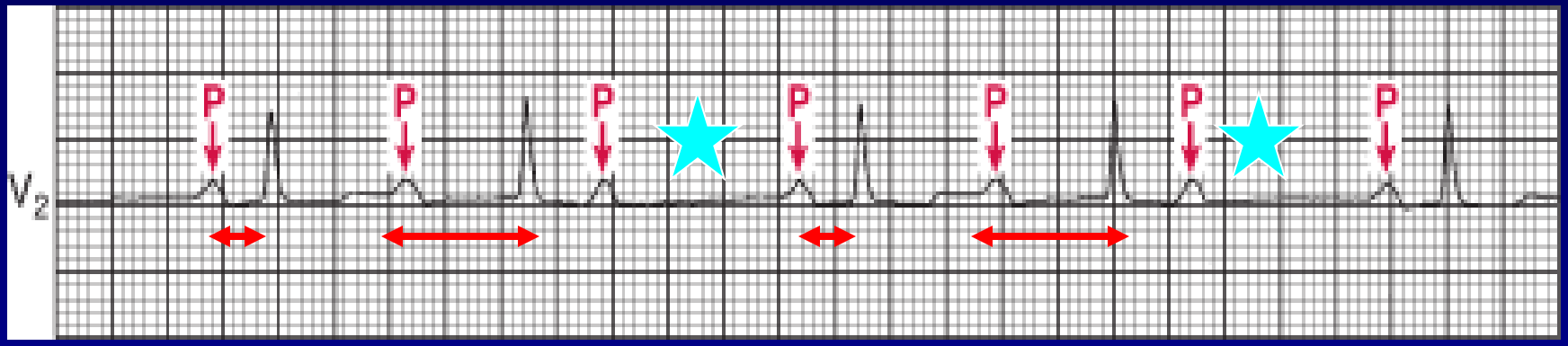
- PR Interval prolongs with each beat until a dropped beat is seen
- The PR Interval is NOT constant
- After each dropped beat, the PR interval is normal and the cycle starts again

Second Degree Heart Block (2º)

Mobitz Type I (Wenkebach)

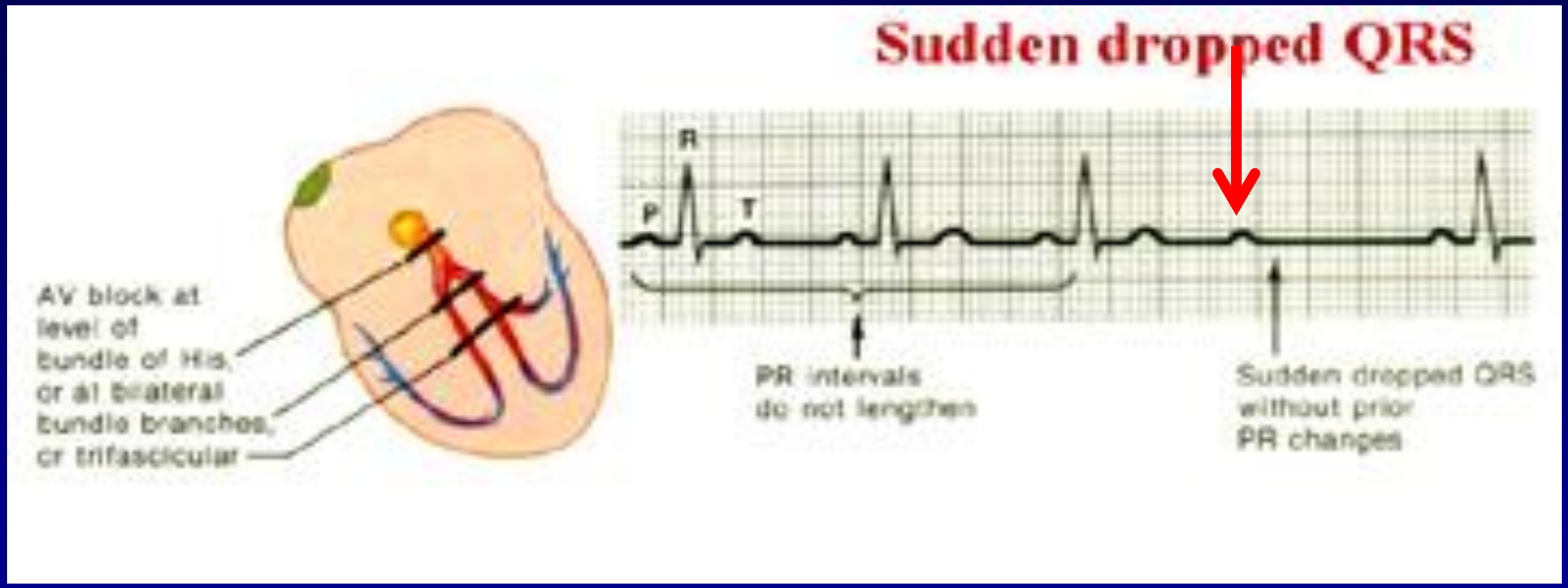


2nd Degree AV block Mobitz 1

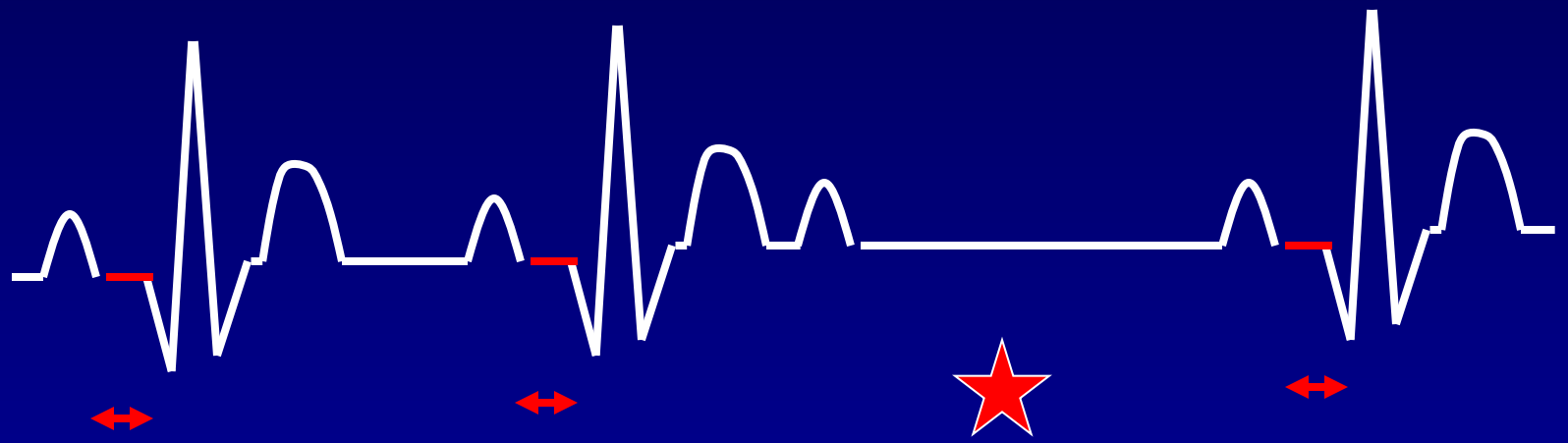


Second Degree Heart Block (2º)
Mobitz Type II

- Conduction through the AV node is constant.
- PR interval is normal and constant
- Occasionally a dropped beat is seen


Sudden dropped QRS
AV block at level of bundle of His, or at bilateral bundle branches, or trifascicular
R
P
T
PR intervals do not lengthen
Sudden dropped QRS without prior PR changes

Second Degree Heart Block (2º) Mobitz Type II



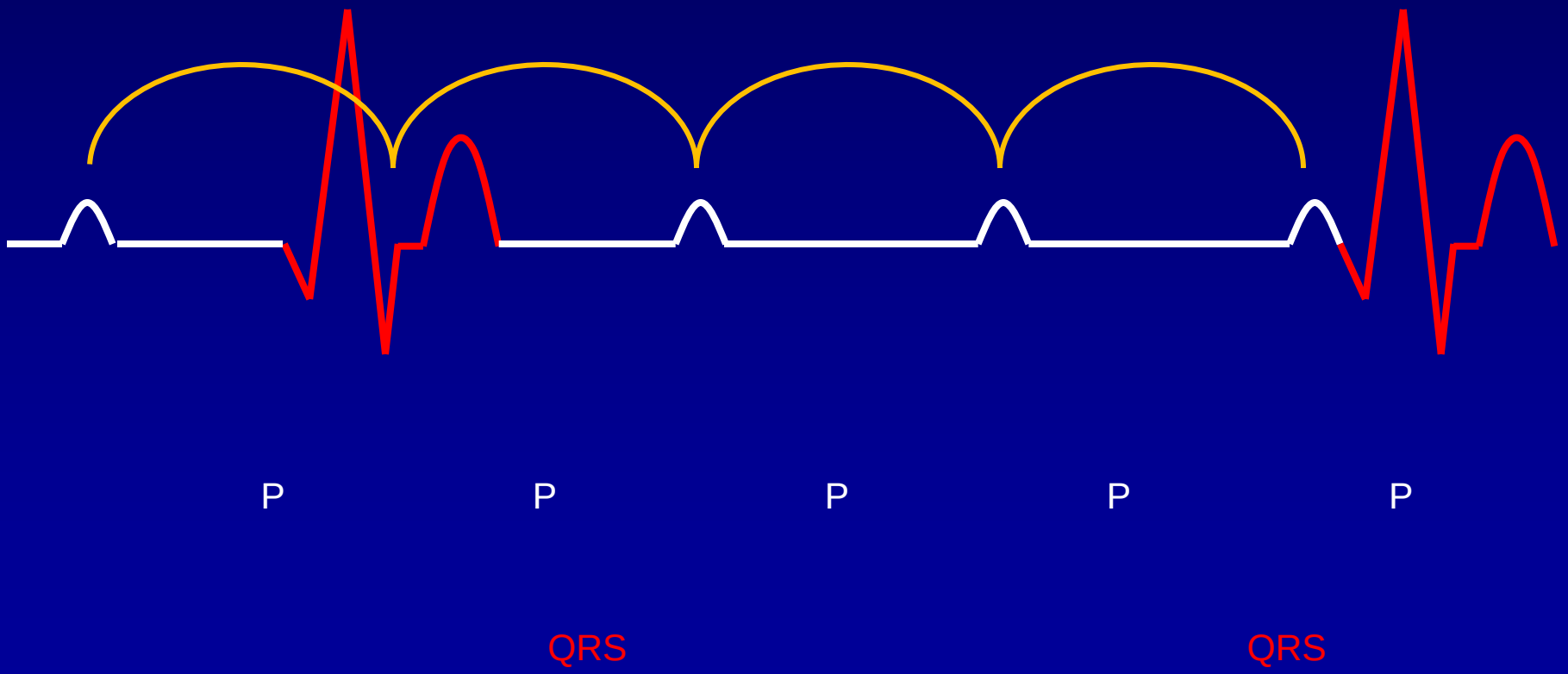
Third Degree Heart Block (3º) (Complete)

- Complete failure of the AV Node
- No impulses from Sinus Node will pass through to the ventricles
- Some part of the conducting system will take over as pacemaker of the heart (even a myocardial cell 10-15 bpm)

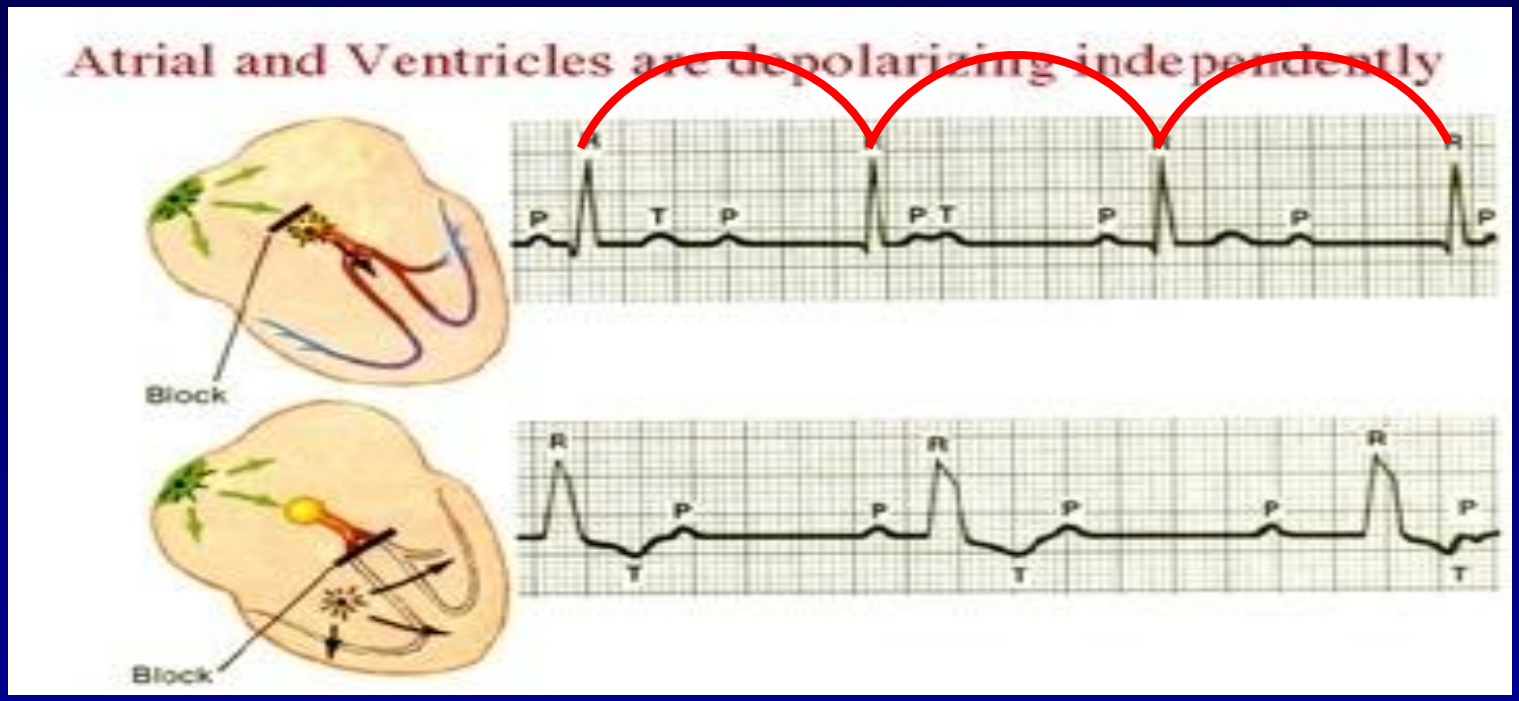
Third Degree Heart Block (3º) (Complete)

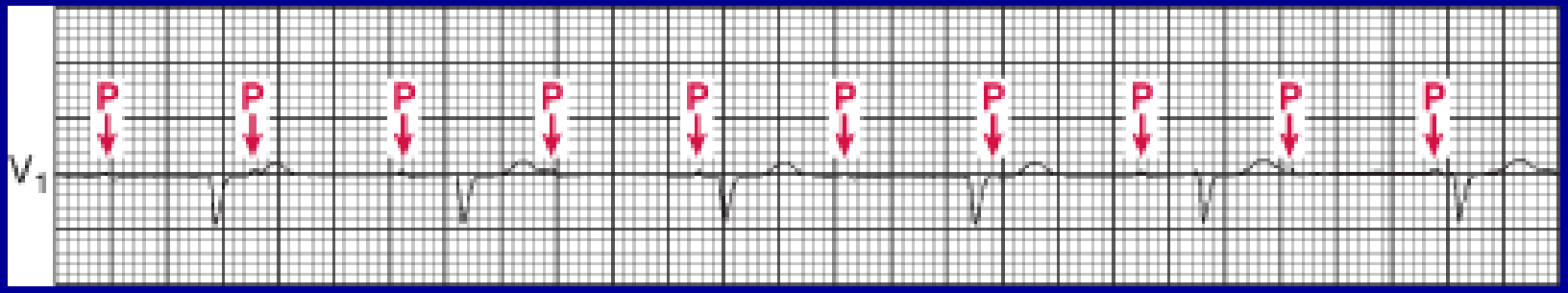
- Independent A and V deplarization
- A rate > V rate
- If V rate > A rate = AV dissociation
- Regular R-R interval.
- Escape rhythm = junctional , ventricular, paced.
- Regular P-P interval

Third Degree Heart Block (3º) (Complete)



3rd degree AV block





Management of AV Block

First Degree Heart Block (1º)

- Interventions include treating the underlying cause and observing for progression to a more advanced AV block.

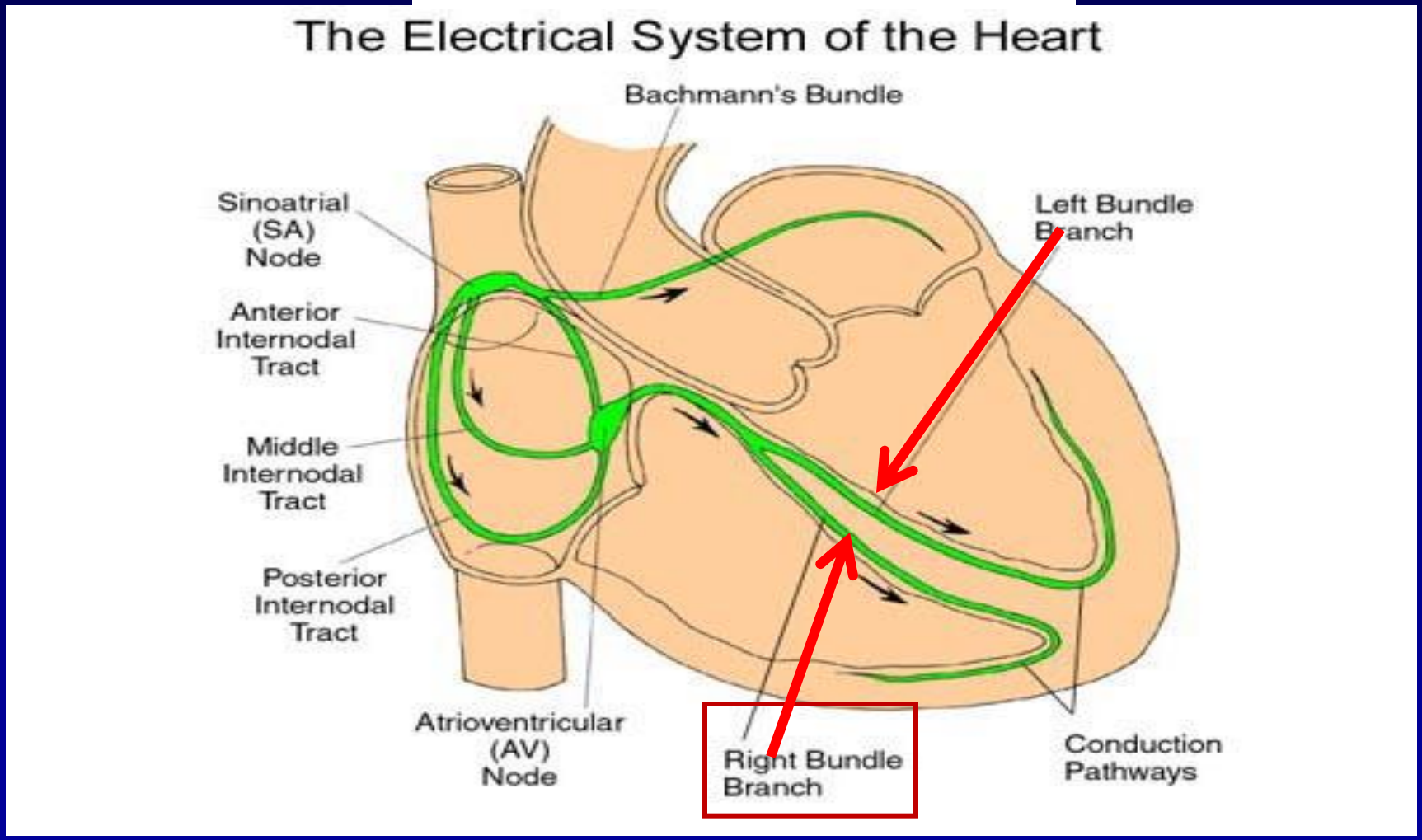
Second Degree Heart Block (2º) Mobitz Type II

- It is more serious than the type I block.
- Treatment is usually artificial pacing.

Third Degree Heart Block (3º) (Complete)

- External pacing and atropine for acute, symptomatic episodes.
- Permanent pacing for chronic complete heart block.

THE CONDUCTION SYSTEM OF THE HEART



Types of BBB

- RBBB
- LBBB

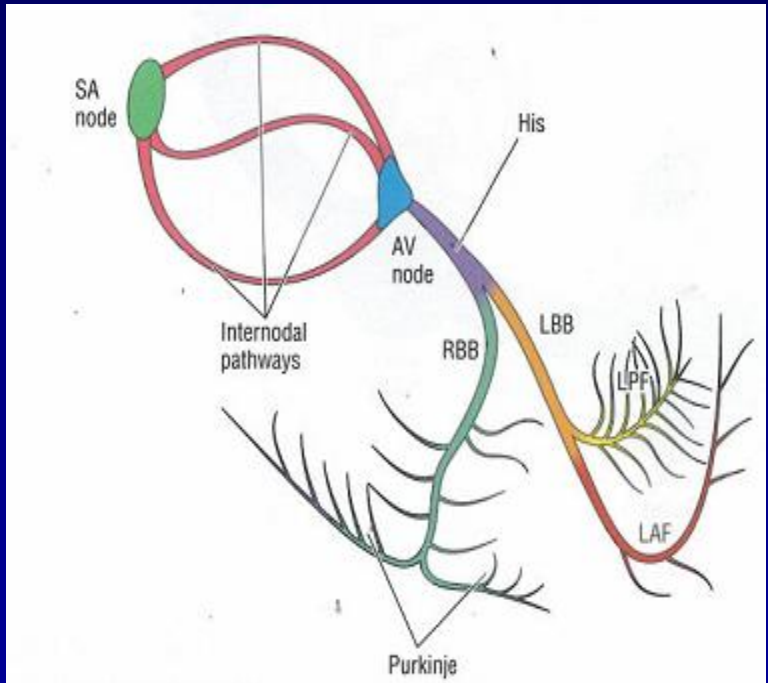


Figure 1-6: The electrical conduction system.

RBBB

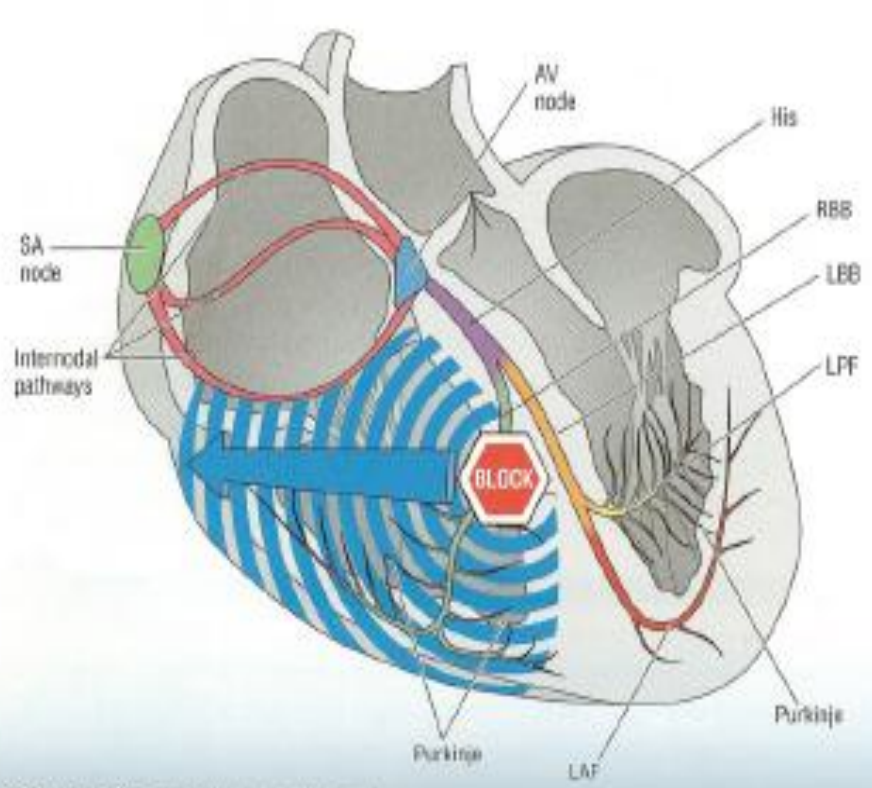


Figure 13-2: Right bundle branch block.

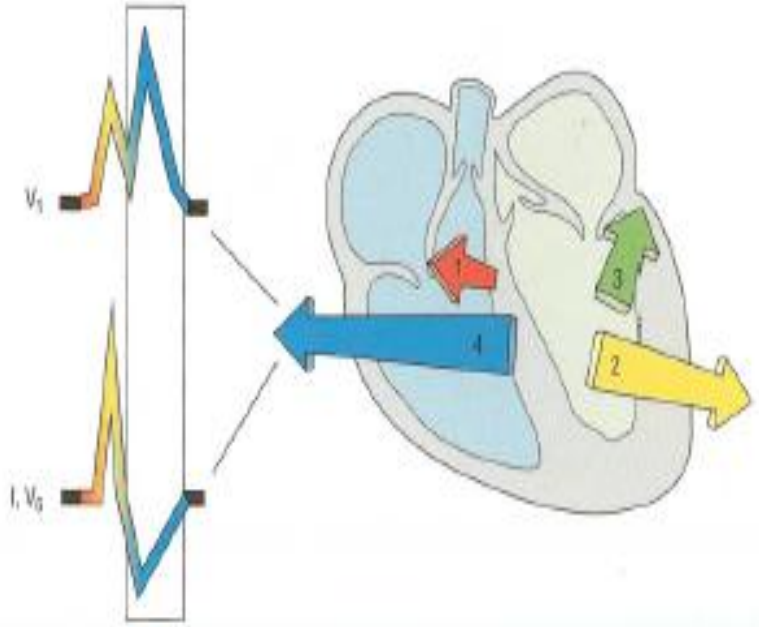
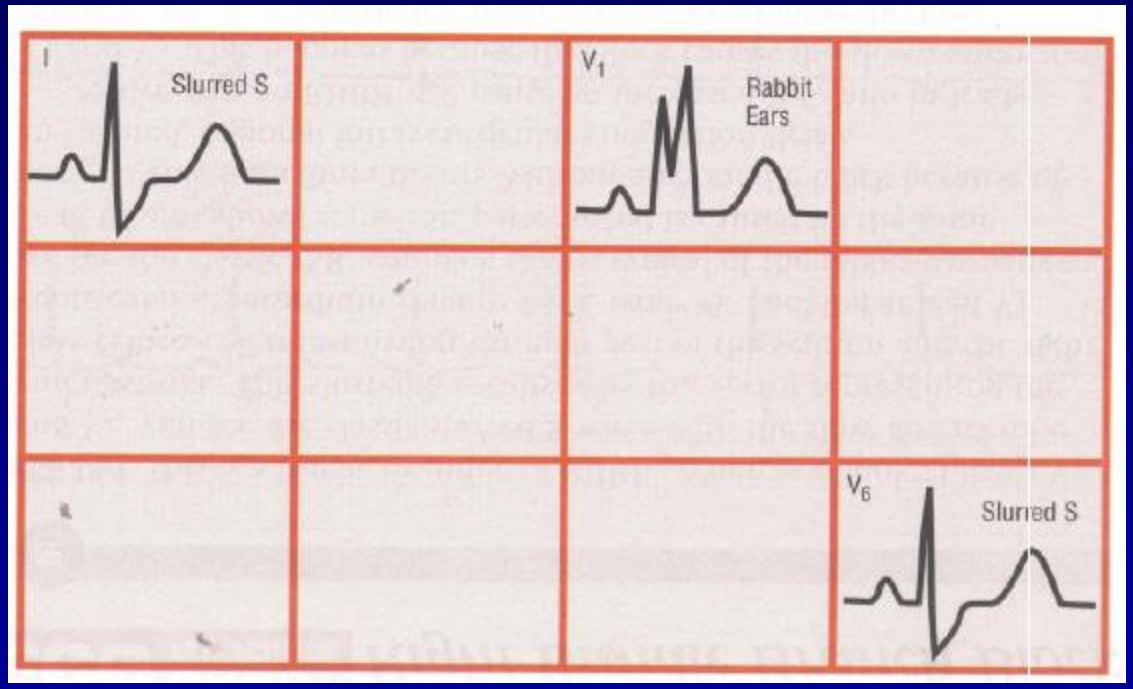


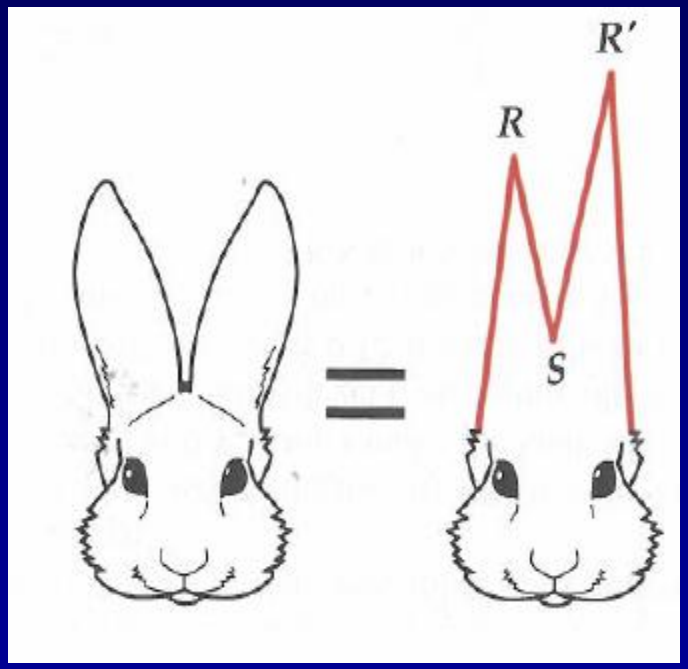
Figure 13-3: RBBB's effect on the ECG.

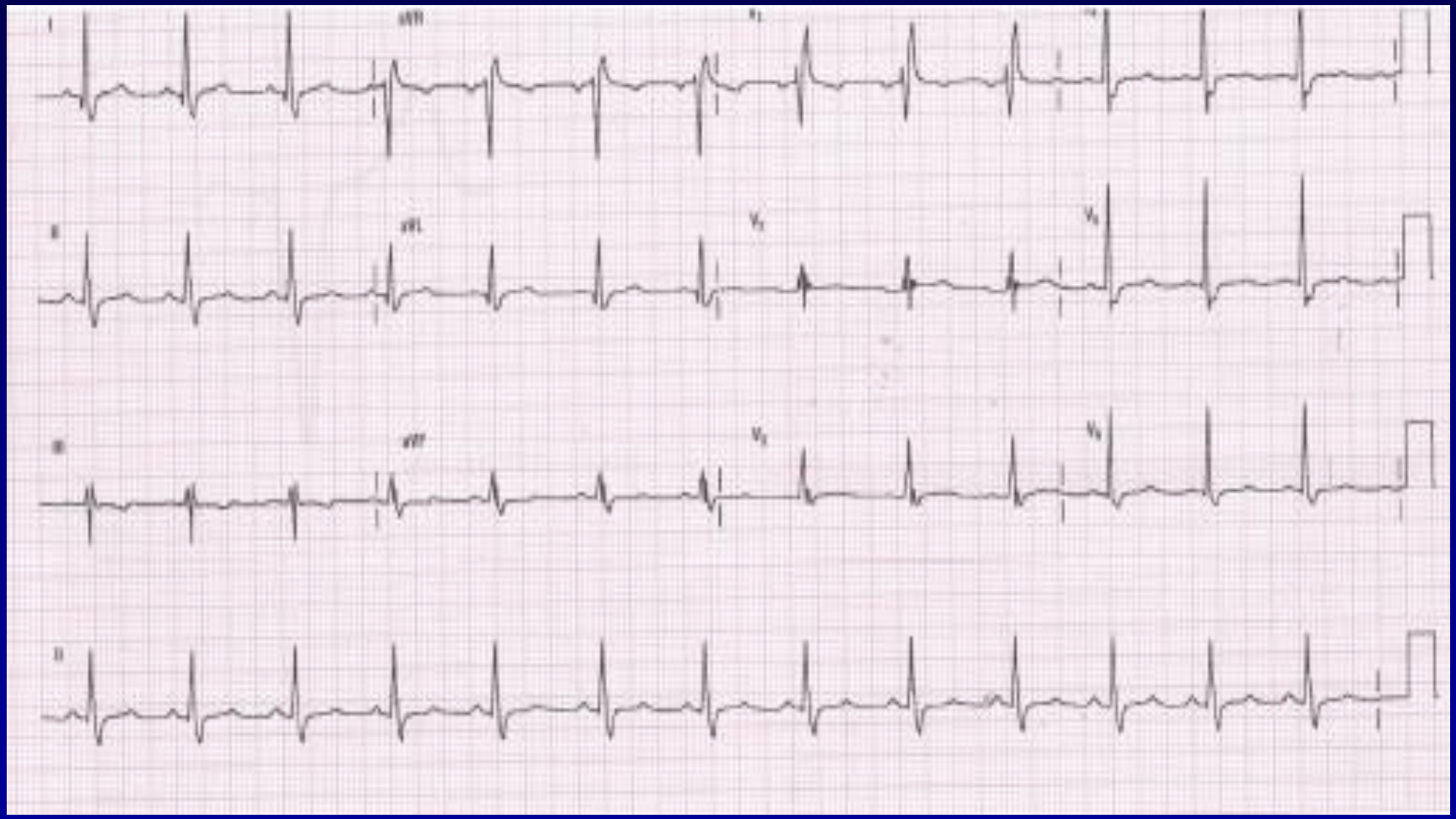
RBBB CRITERIA

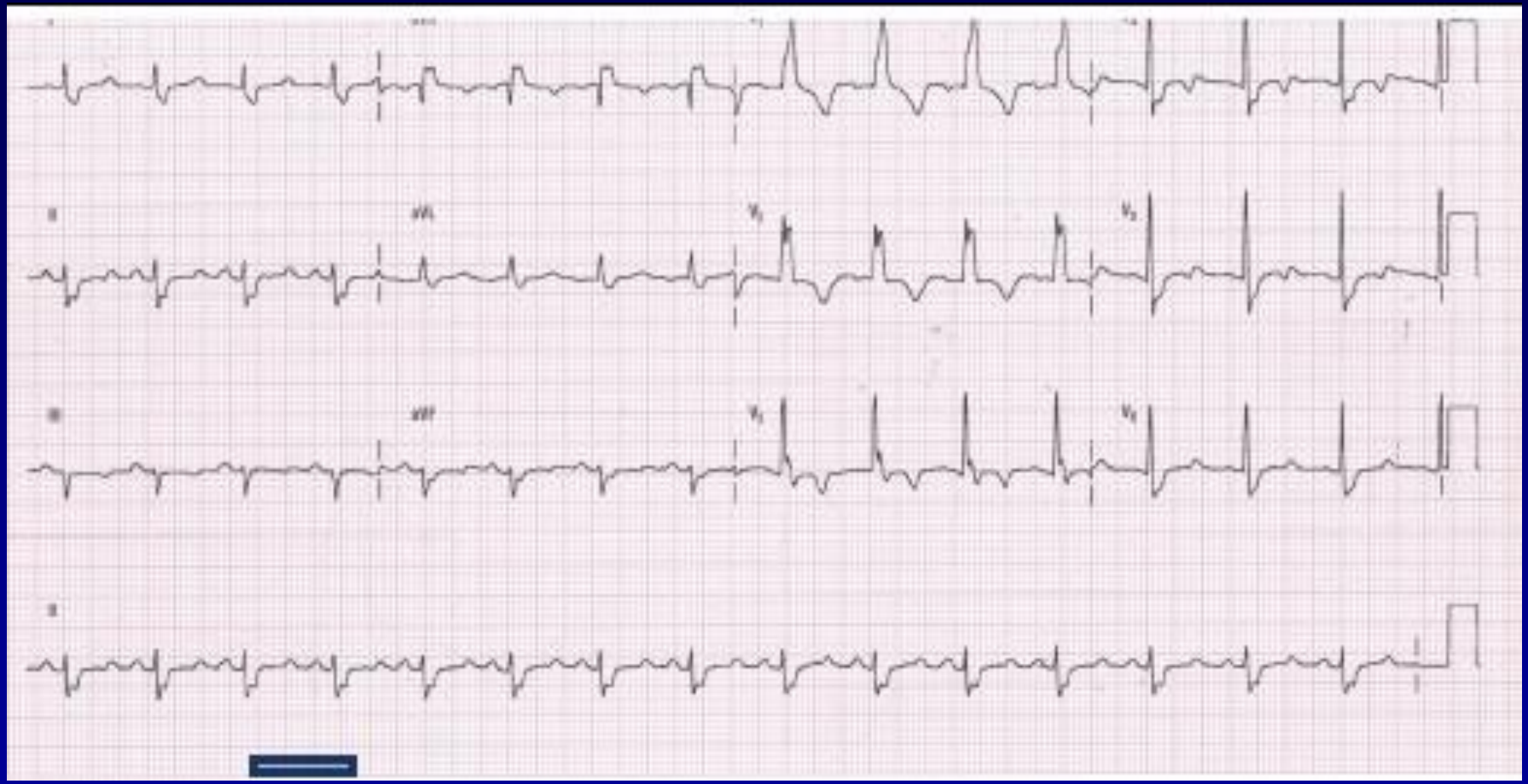
- Prolonged QRS duration (> or = 0.12 seconds)
- Secondary R wave (R’) in leads V1 and V2 (rsR’ or rSR’)
- Secondary ST & T-wave changes (T wave inversion; downsloping ST segment may or may not be present) in leads V1 and V2
- Wide slurred S wave in leads I, V5, and V6

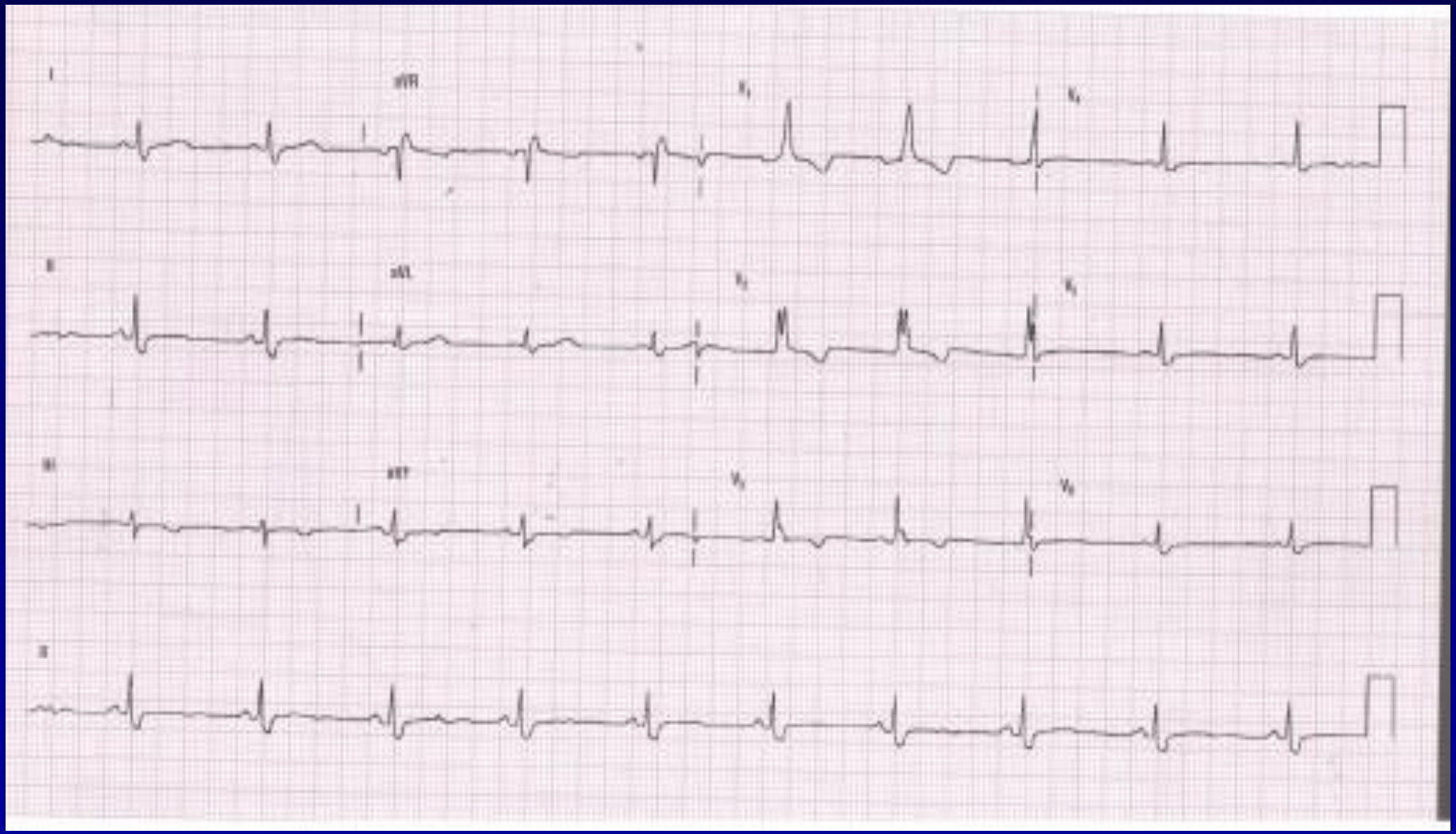
RBBB CRITERIA



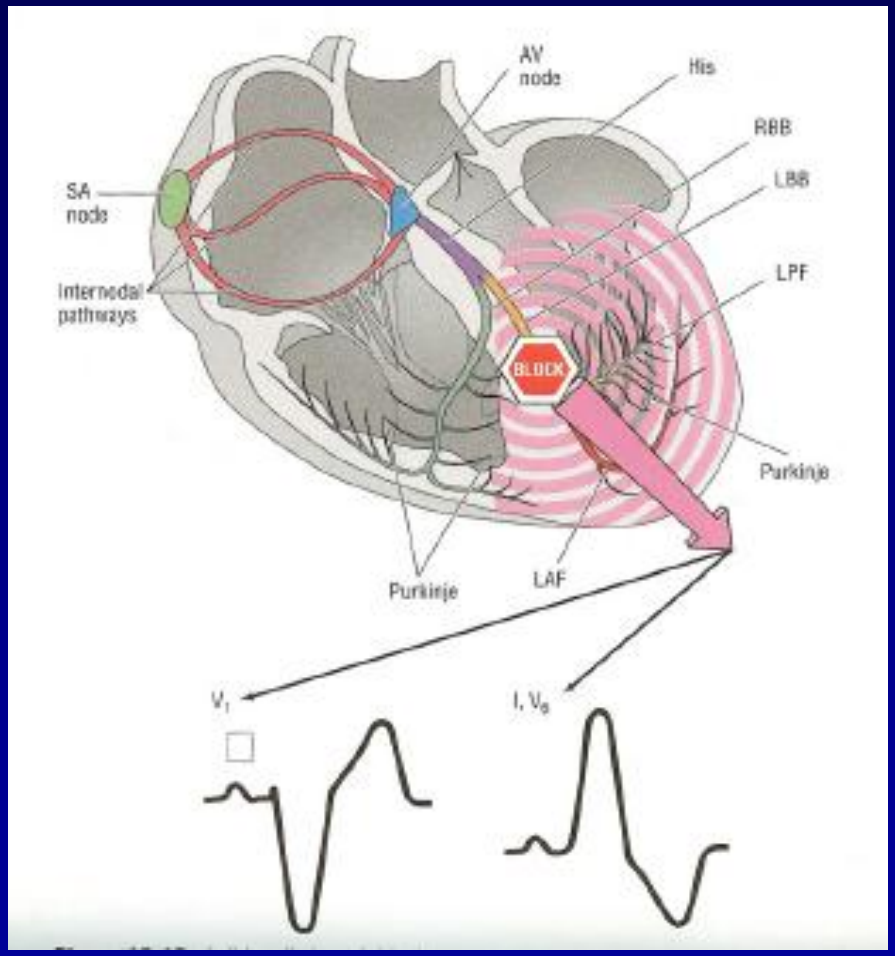


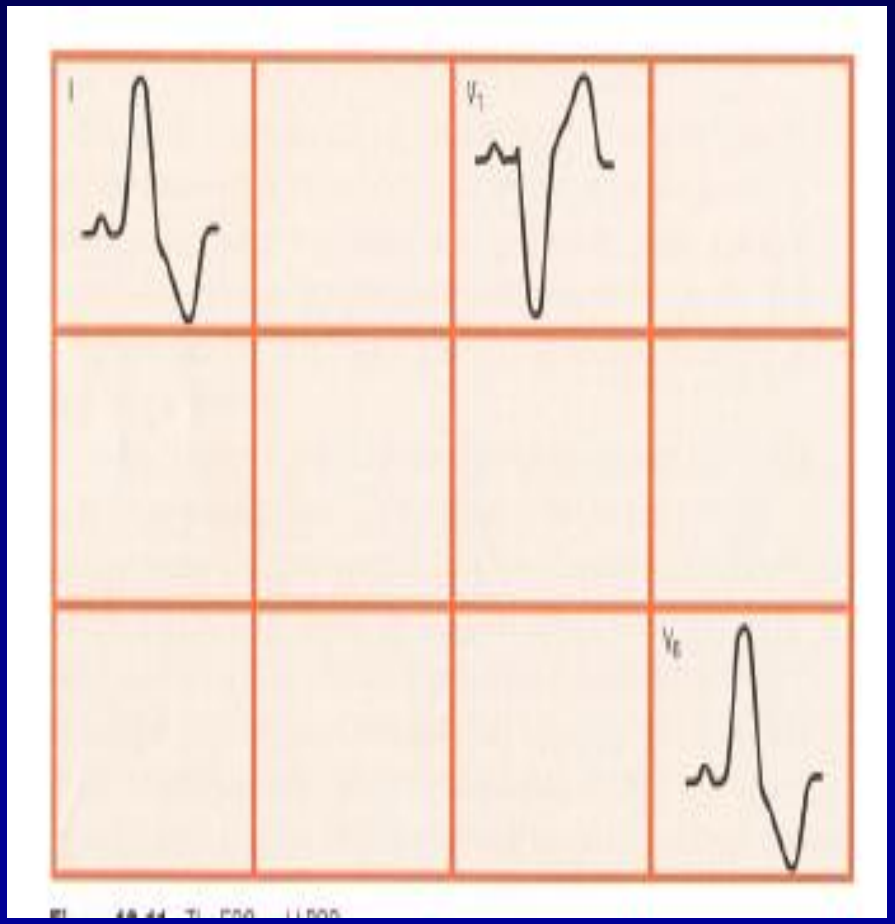




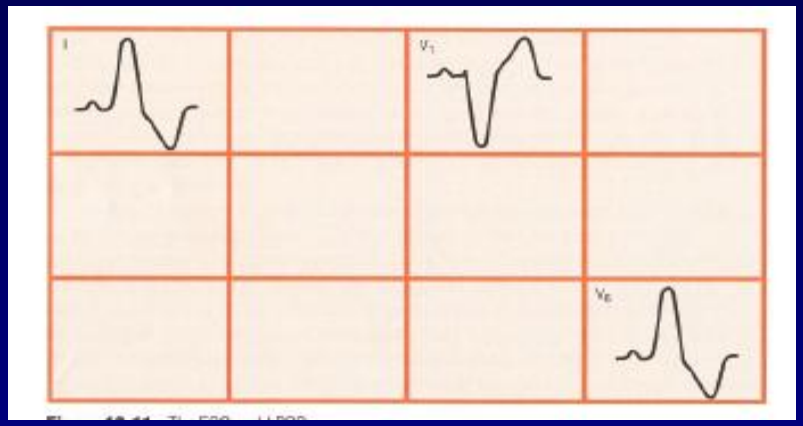


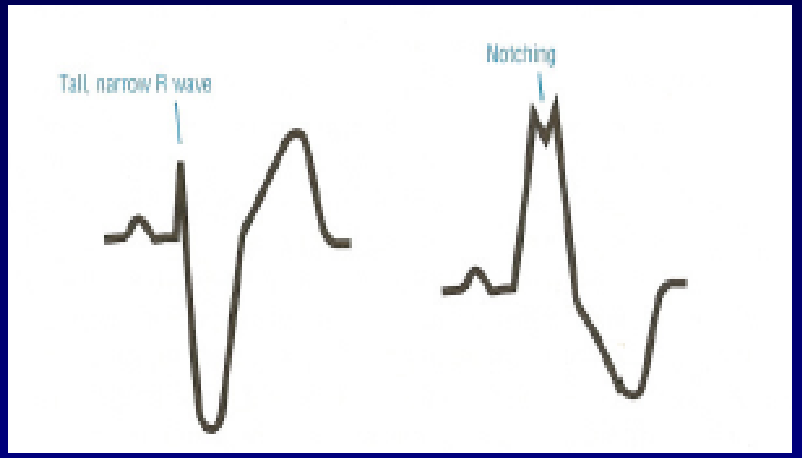
LBBB CRITERIA



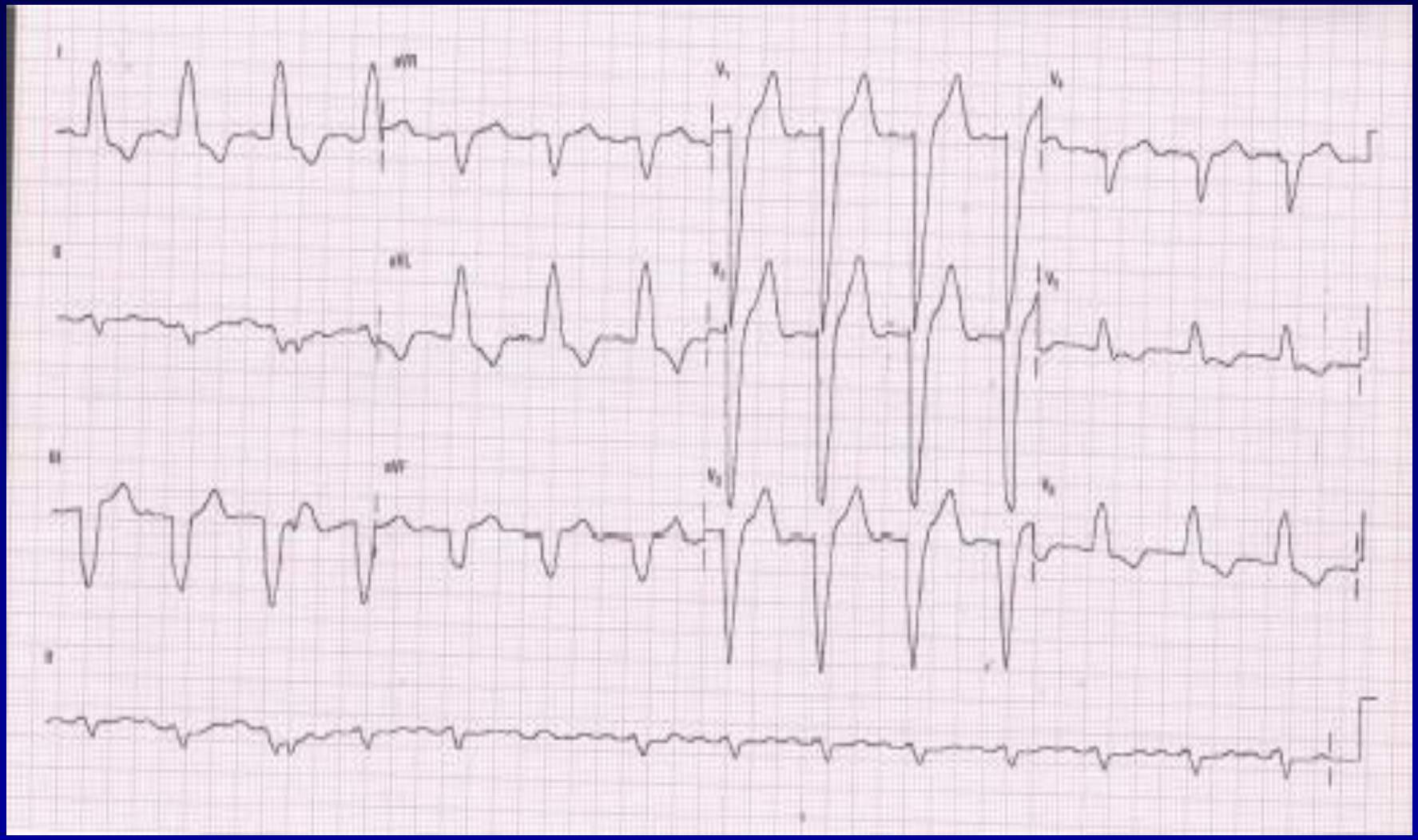


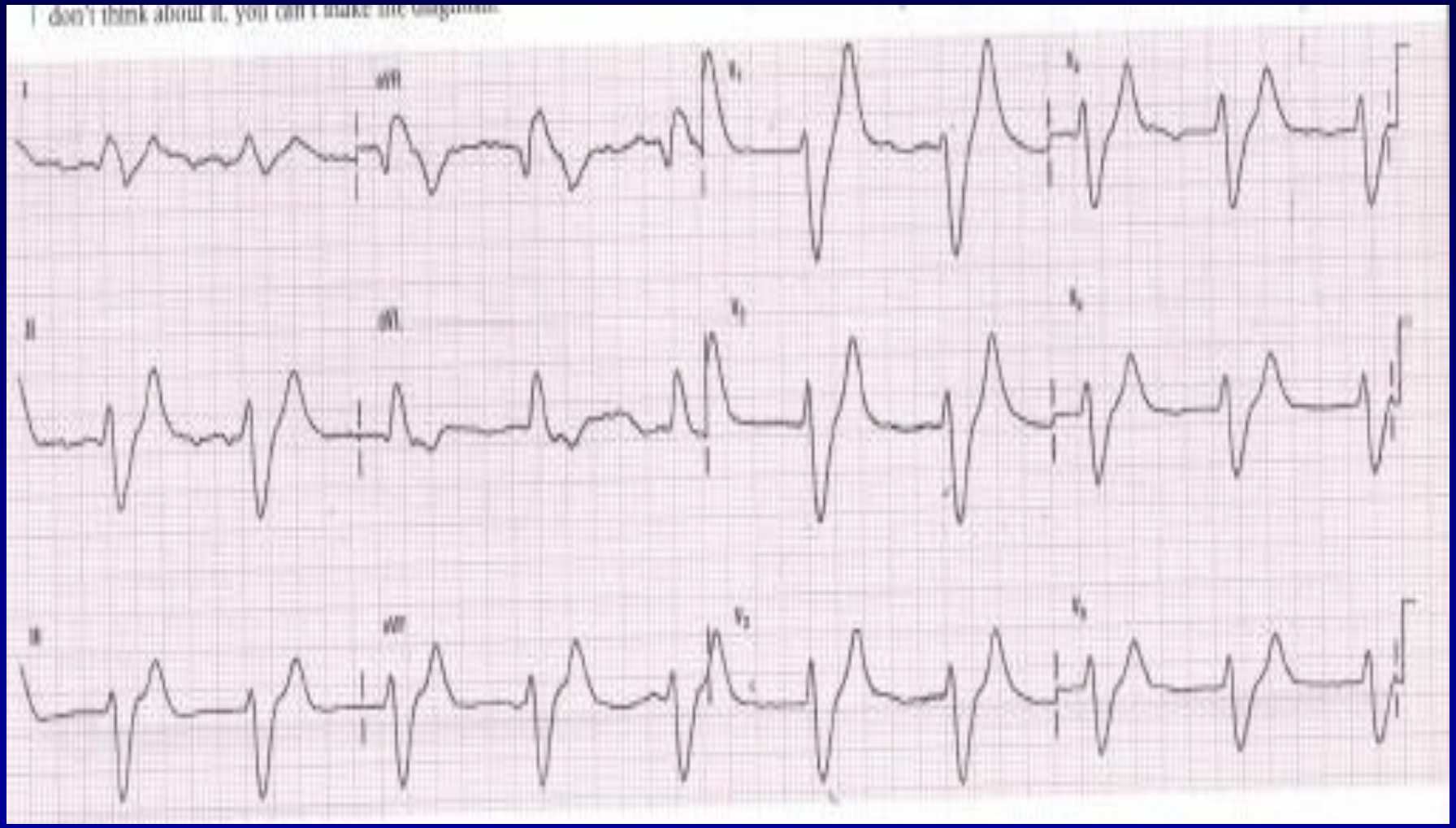
LBBB CRITERIA

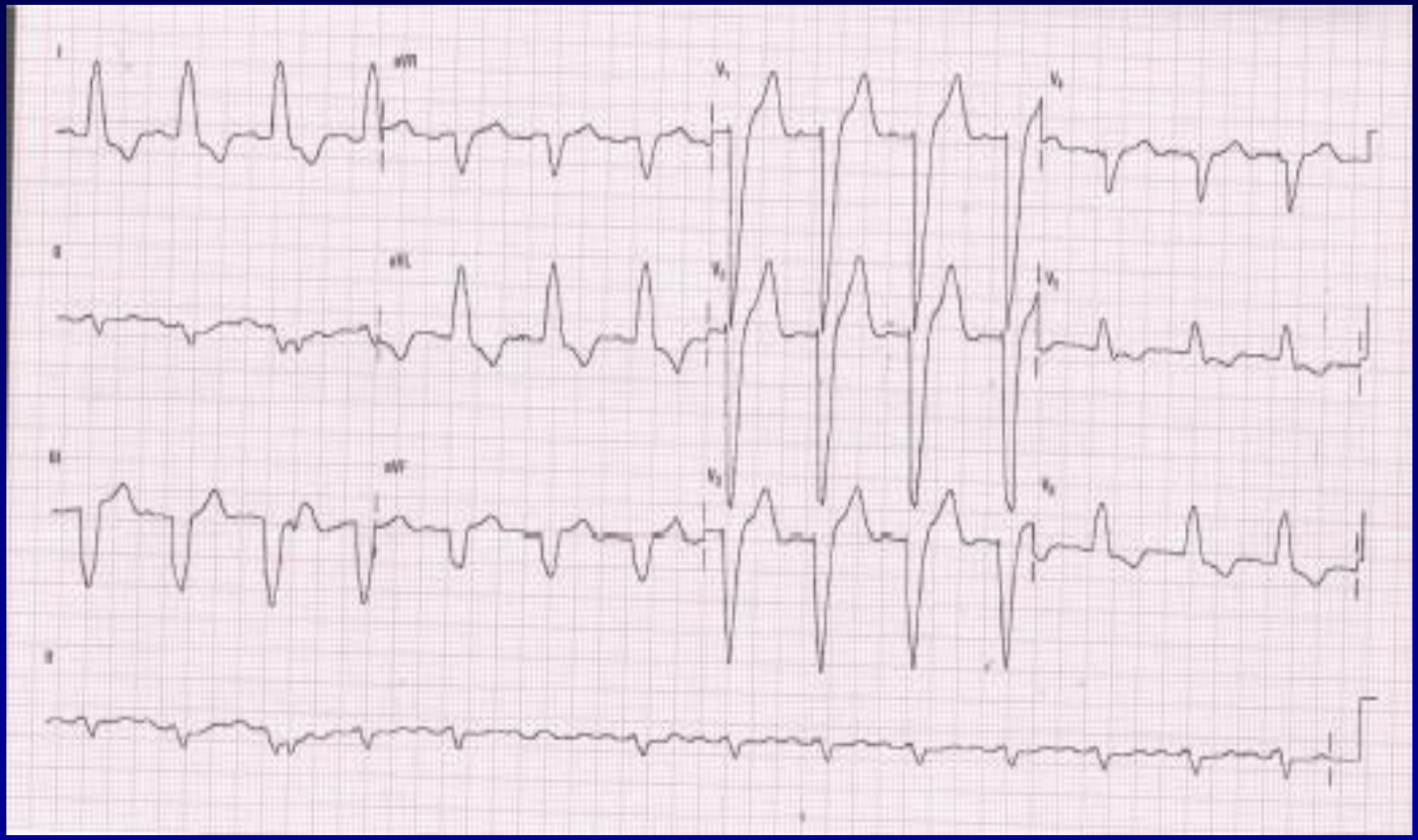




- Prolonged QRS duration (>0.9 - 0.12 seconds)
- **Broad monophasic R waves in leads I, V5, V6 that are usually notched or slurred**
- Secondary ST & T wave changes opposite in direction to the major QRS deflection (i.e., ST depression & T wave inversion in leads I, V5, V6; ST elevation & upright T wave in leads V1 and V2)
- rS or QS complex in right precordial leads







RBBB and LBBB

Left bundle branch block is more ominous than right bundle branch block because it usually is present in diseased hearts.

Both may be caused by:

- Hypertension,
- MI,
- Cardiomyopathy.

A Bifasicular block may progress to third degree heart block. **Treatment is artificial pacing** for a bifasicular block that is associated with an acute MI.

Thank you